The American Journal of Surgery

NEW SERIES, VOL. XIV DECEMBER, 1931 No. 3

DIAGNOSTIC AND THERAPEUTIC INJECTIONS OF THE SYMPATHETIC NERVES*

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THE two greatest objections to therapeutic measures directed toward the sympathetic nervous system have been the uncertainty and experimental nature of operative results, and the magnitude of the surgery.

The first objection has been overcome by the introduction of diagnostic injections to determine operability. The second objection still holds, but therapeutic injection of alcohol may be used in many instances to replace surgery.

DIAGNOSTIC INJECTIONS

Since the work of Brown,[1] White[2] and Mixter, Scott and Morton,[3] Flothow,[4] and others, on injection methods for diagnosis, operative procedures should not be undertaken without a preliminary diagnostic test; the only exceptions to this statement occur in the case of Hirschsprung's disease and in spastic conditions, where the time element plays an important part. Even in these conditions preliminary alcohol injection offers several months' ablation of the sympathetics in which to judge potential results from operating. This is particularly true in spastic conditions; the operative results in Hirschsprung's disease are so constant that diagnostic injections are not necessary.

There are several diagnostic injection methods available. The original fever test, introduced by Brown, consists in the intravenous injection of typhoid vaccine to produce vasodilatation. There are several objections to this method: it causes a severe generalized reaction with nausea, vomiting, and extreme discomfort; it is an indirect test, producing only vasodilatation and does not give any information as to relief of pain. We feel that this test is the least reliable of any.

Spinal anesthesia is a simple method of producing vasodilatation, operability being determined by the degree of increase in temperature. This test is available only for the lower extremities and gives no indication as to pain relief since all pain fibers are anesthetized.

Direct injection of nerve trunks is effective but incomplete, as all sympathetic fibers are not interrupted.

The method which we prefer and use almost entirely is the direct injection of sympathetic nerves, the technique being simple and causing the patient very little discomfort. By anesthetizing at definite levels we can determine accurately what operative removal would accomplish. Its great advantage is in relation to pain, for if pain is relieved, either removal or alcohol injection will produce the same result. This is important in cases in which it is possible to relieve pain even though vascularity may be unimproved.

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Read before the Association of Residents and Ex-Resident Physicians of The Mayo Clinic, Rochester, Minnesota, October 10, 1931.

TECHNIQUE OF INJECTIONS

Lundy's[5] method of injection is preferred since it is uniform for all portions of the sympathetic chain. It is done in the prone position with the patient lying upon the abdomen, properly supported by pillows.

Labat's[6] technique for paravertebral injection may also be used. In the dorsal region, however, one is more apt to puncture the pleura or the dura than in Lundy's method. For lumbar injections the Labat technique is an alternative if one is unable to strike the sympathetic chain by Lundy's technique.

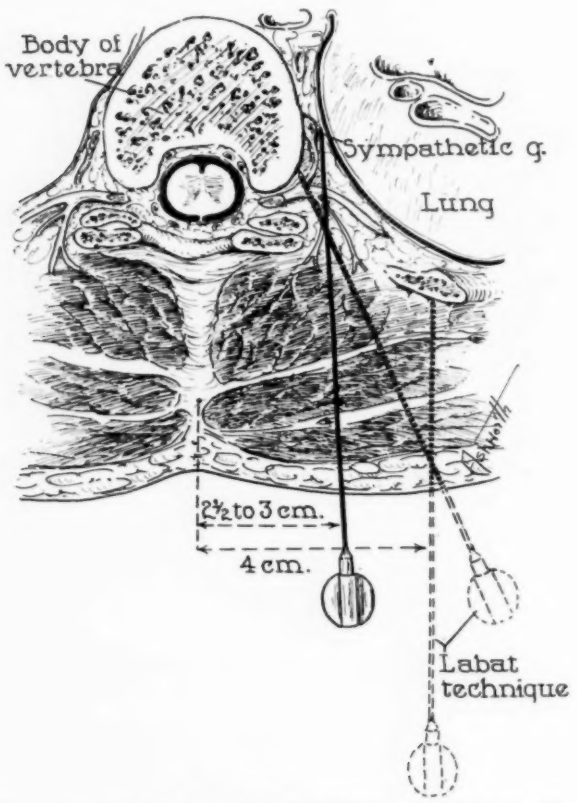

FIG. 1. Point of injection of needles in dorsal region. Showing Lundy method and Labar technique. (From Labat.)

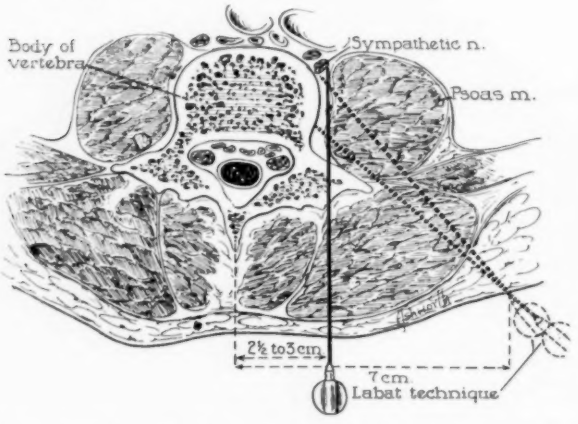

FIG. 2. Illustrating Lundy and Labat technique for lumbar injection. (From Labat.)

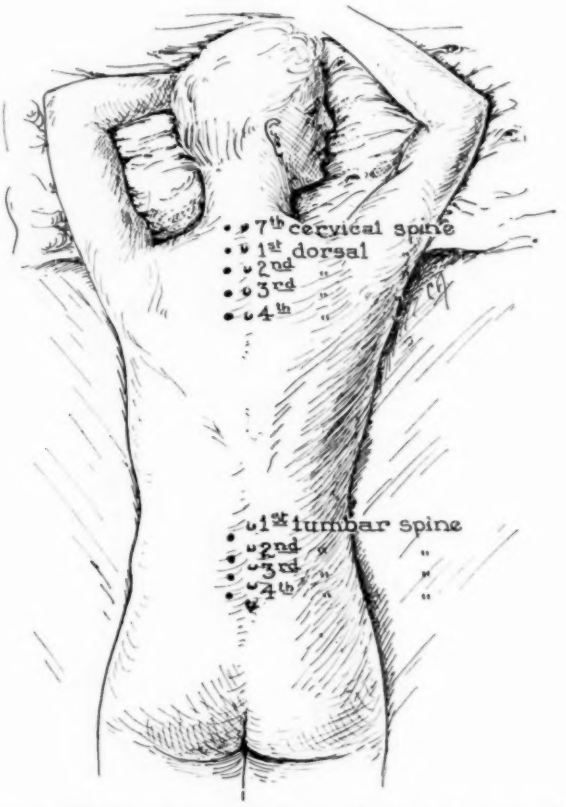

FIG. 3. Point of insertion of needles for dorsal and lumbar injections using Lundy technique. First dorsal segment lying opposite the seventh cervical spine, showing that dorsal injection is made opposite spine and lumbar injection opposite interspaces.

In Lundy's method the point of insertion of the needles is approximately two fingers' breadth lateral to the midline, opposite the spines in the dorsal region, and opposite the interspaces in the lumbar region. The actual distance varies from 2½ to 3 cm., depending upon the size of the bodies of the vertebrae, the object being to skirt the edge of the bodies closely in inserting the needles. The insertion is made perpendicularly until the transverse process is encountered. The needle is then angled so as to slide either just above or just below the transverse process, and then inserted two fingers' breadths deeper,

again from 2½ to 3 cm., depending upon the size of the individual. Five cubic centimeters of one per cent procaine are deposited into each needle and if the needles are properly placed, increased temperature of the extremity will be apparent in from five to fifteen minutes, depending upon how close to the sympathetic chain the points of the needles lie. If no temperature increase occurs within fifteen minutes, the needles should be withdrawn and reinserted either closer to the bodies of the vertebrae or at a slight angle toward the midline.

Two common reasons for failure are placing the point of the needle too far laterally or not deep enough. Failures are rare in the dorsal region since the sympathetic chain lies lateral to the bodies of the vertebrae. They are more common in the lumbar region where the chain lies more on the anterolateral aspect of the bodies of the vertebra or anteriorly. In such instances the Labat approach for paravertebral injection can be employed, starting from a point 7 cm. from the midline and inserting the needle at an angle of 45°, then angling until it slides over the anterolateral surface of the body of the vertebra.

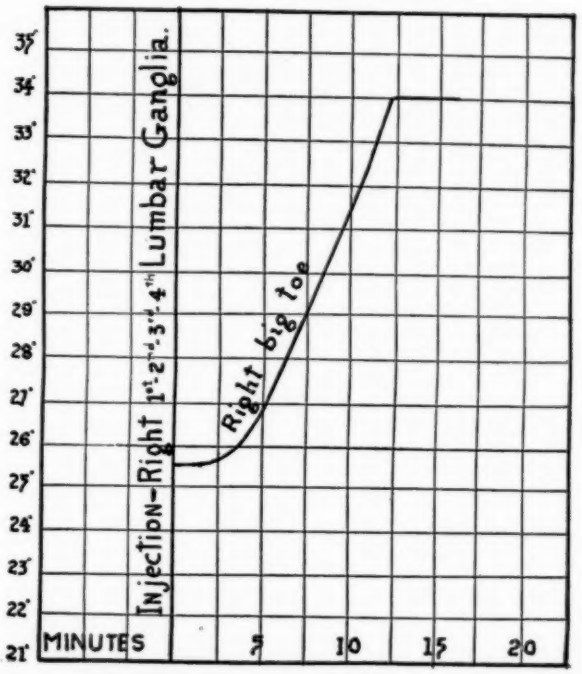

Fig. 4. H. E. Aged forty-seven. Diagnosis: (1) arteriosclerotic endarteritis; (2) Buerger's disease. Complaint: cold, painful extremities. Increase of 8.3°C. showing marked element of vasospasm. Complete relief of pain. Injection of alcohol resulted in complete relief with same increase in temperature.

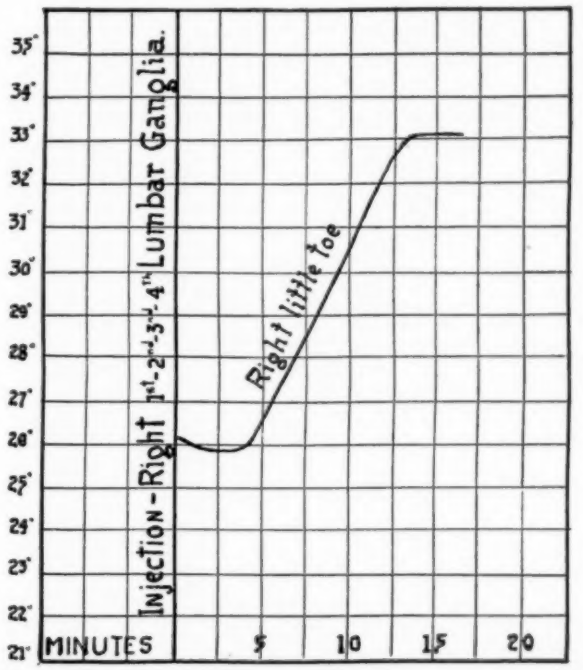

Fig. 5. V. J. Aged fifty-one. Diagnosis: arteriosclerotic endarteritis. Date: May 20, 1931. Complaint: cold, cyanotic feet, claudication. Increase of 7.0°C. Foot became warm and pink. Pain completely relieved. Alcohol injected.

The procedure for diagnostic injection is as follows. The test should be done in a cool room, preferably about 60°F., since in a hot room, where vasodilatation may be present, results would be misleading. The extremities to be tested are exposed for at least one-half hour to the room temperature before skin temperature readings are taken. In order to assure accuracy it is essential that some form of thermocouple be used, and for these readings we use a dermatherm. The needles are then inserted as follows: for conditions above the clavicles, the needles are placed opposite the seventh cervical, first and second dorsal spines; for the arms, opposite the first, second and third dorsal spines; for

the legs, opposite the first, second, third and fourth lumbar interspaces. When the needles are in place 5 cm. of 1 per cent

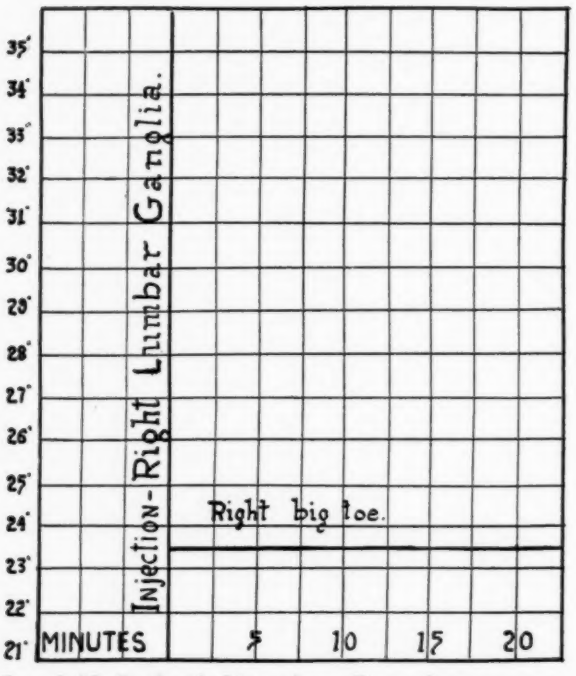

Fig. 6. H. R. Aged thirty-three. Date: Oct. 10, 1930. Complaint: Gangrene of right big toe. Severe pain. Diagnosis: Burger's disease. Result: no increase in temperature of toe. Leg warmed up to sharply limited area just below knee, from there down remained cold. Advised amputation above knee. Done.

procaine are deposited in each needle. Temperature readings are then taken at five minute intervals for twenty minutes, always using the uninjected extremity as a control. Pain may disappear before the vasomotor changes are apparent. We feel that a rise of at least 5°C. is necessary to indicate operation in conditions due to vascular insufficiency such as Buerger's disease and Raynaud's disease. Morton and Scott[3] have given us a minimum vasodilatation level at 31.5°C. which is very important. Obviously, if the room is very warm with vasodilatation present before injection, there may be very little rise in skin temperature and therefore allowance should be made for the temperature of the room. If pain is relieved and the dermatherm readings show increase of from 5° to 10°C. in temperature, either alcohol injection or operation is indicated.

COMMENTS

One of the first cases in which we employed this diagnostic injection was a girl

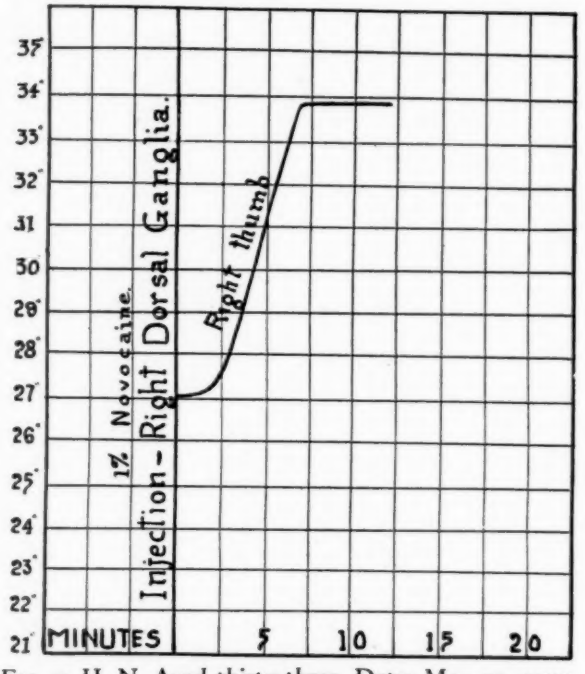

Fig. 7. H. N. Aged thirty-three. Date: May 13, 1930. Complaint: cold, blue painful extremities. Diagnosis: Raynaud's disease. Increase of 6.7°C., showing marked vasospasm. Cold, blue, cyanotic hand changed to warm, pink one. Injection of alcohol.

twenty years of age, who, following an injury, developed a severe pain in her hand associated with bluish discoloration and excessive cold perspiration. Novocaine injection of the upper dorsal segments produced complete relief of pain, marked increase in temperature, and changed the blue, cold moist hand to one warm, pink and dry. Removal of the sympathetic ganglia in this case gave complete and lasting relief. This case has been reported in detail.[7]

To differentiate atypical facial pains it is only necessary to inject the inferior cervical and upper two dorsal segments. If pain is relieved, one may be sure that it is sympathetic in origin or transmission.

In thromboangiitis obliterans (Buerger's disease) operability is easily determined. A marked increase in temperature and relief of pain assures good results following operation or alcohol injection. If this

does not occur, amputation is indicated, and the diagnostic injection will give a definite level for amputation. If alcohol is injected it insures maximum blood supply and should lower the incidence of painful stump.

In Raynaud's disease the same holds true. The operative result is reproduced preoperatively by injection of the proper sympathetics. In scleroderma, so often associated with Raynaud's disease, the amount of temperature increase following injection is the criterion of operability.

In chronic arthritis, definite information may be obtained from the diagnostic injection. We have had several cases in which relief of pain, marked increase in joint function, and marked temperature increase followed injection. Operation in these cases reproduced those results.

In arteriosclerotic pain and gangrene, diagnostic injection is invaluable as many of these patients can be spared amputation. We shall take up this condition more fully in the discussion on therapeutic injections.

In trophic lesions of extremities, skin lesions, causalgias, intermittent claudication, painful stump, diabetic neuritis, and various other painful conditions of unknown origin, valuable information may be obtained from diagnostic injections which indicate therapeutic measures of proved worth.

To summarize: diagnostic procaine injections of the sympathetic nerves at various levels give us definite indications for successful treatment in a great variety of conditions. These injections have taken the guess work entirely out of the realm of sympathetic therapeutic measures.

THERAPEUTIC INJECTIONS

The scope of this paper is too great to allow a comprehensive discussion of every phase. The past two years have contributed a new therapeutic measure of great value in the alcohol injection of sympathetic nerves. We now have a method which has in many instances replaced the highly specialized operation of sympathetic ganglionectomy. It has made possible the successful treatment of a wide variety of conditions in which, owing to the risk, the magnitude of the operation, or the economic status of the patient, operation was not justified.

The substitution of injection for operation is not advocated except where operation is not warranted. We frequently meet borderline cases in which the diagnostic injection shows an uncertainty as to results from operation which would justify its use. In these cases one can use the injection of alcohol not only as a therapeutic measure but also as a glorified diagnostic procedure, the results of which may warrant operation later. Alcohol injection is reserved for those cases in which we feel that operation is contraindicated. We have done approximately fifty alcohol injections for a variety of conditions with excellent results.

In conditions such as Buerger's disease, Raynaud's disease, scleroderma, chronic arthritis, Hirschsprung's disease and spastic conditions where a complete and lasting denervation is essential, we remove the sympathetics surgically in suitable cases.

ARTERIOSCLEROTIC CONDITIONS

Since our recent report[8] of the results of alcohol injections in 8 cases of arteriosclerotic pain and trophic conditions, 4 more have been done. The immediate results have been excellent to date, although the time elapsed has been too short to determine the lasting qualities of this treatment. It would seem that the pathologic process of this condition might preclude expectation of success, but, strangely enough, our results have been as good or better than in those conditions previously considered suitable for surgery or injection.

Alcohol injection is preceded as usual by diagnostic procaine injection. It is astonishing to find these patients obtaining almost instant relief of pain and marked improvement in circulation when procaine is injected. If this occurs, 5 to 7 c.c. of 95 per cent alcohol is injected in each needle and

gives permanency to the result. We have had 12 cases, 10 of which resulted in complete relief of pain and healing of trophic lesions. In one case gangrene was already present up to the ankle, and injection was done merely as a measure to promote maximum blood supply to the stump, amputation being considered urgent. Following the injection which preceded amputation by three days, there was an immediate demarcation and the marked edema which had been present had entirely disappeared. The other case was a complete failure, amputation disclosing complete obliteration of all vessels. It is obvious that if vessels are completely obliterated one cannot hope for improvement.

The injection in these cases is one of the most satisfactory phases of all our work, since previous to our discovery of the treatment, very little short of amputation gave relief.

In all cases where amputation is necessary due to vascular deficiency of any nature, amputation should be preceded by alcohol injection of the sympathetics to insure maximum blood supply to the stump, thus lessening the incidence of non-healing stump and insuring against painful stump. In a case of arteriosclerotic gangrene alcohol injection was done immediately preceding amputation, performed at an astonishingly low level in the face of evident very poor blood supply, healing resulting by primary intention.

In this group of cases injection is preferable to surgery owing to the age of the individual and the uncertainty of final results. It is probable that this treatment is only palliative for the process will probably extend and later necessitate amputation which, however, may be delayed several years as a result of the injection.

THROMBOANGIITIS OBLITERANS

We have not as yet used this method in a frank case. Some of the cases classified as arteriosclerotic in nature may have been thromboangiitis obliterans, since it seems impossible to differentiate the two conditions in individuals over fifty years of age and we therefore classify these as arteriosclerotics.

Injection, rather than operation, should be employed in all cases where diagnostic methods indicate that operative results would be questionable or useless. Even where amputation is necessary it insures maximum dilatation of patent blood vessels and therefore maximum blood supply to the stump. When the case is a borderline one as to operability, alcohol injection will often produce such marked improvement as to warrant surgery.

RAYNAUD'S DISEASE

In a case of Raynaud's disease we employed alcohol injection of the sympathetics to the lower extremities as a palliative measure, because the patient, convalescing from bilateral dorsal ganglionectomy, did not wish to undergo another major operation for several months. The results of the injection to date are comparable to those of surgical removal.

CHRONIC POLYARTHRITIS

In 3 cases we have been running a comparative test, ganglionectomy being performed on one side, alcohol injection on the other, both in the upper and lower extremities. To the present time our results have been about the same as to relief of pain and improvement in joint function. As to increased vascularity, however, we have found that the operated extremities are maintaining a higher temperature than the injected ones and it is probable that operation will become necessary on the injected ganglia.

Patients uniformly prefer the operation to the dorsal injection because of the neuritis which follows the injection. This subject will be discussed more fully later.

TRIFACIAL NEURALGIA AND ATYPICAL FACIAL NEURALGIAS

In a case of typical tic douloureux, alcohol injection of the inferior cervical and upper two dorsal ganglia was done

with complete relief of pain for the six months in which we were able to follow the case. The patient had refused to have the sensory root sectioned after direct injection of the nerve failed to bring relief.

In a case of atypical facial neuralgia associated with pain in the neck, arm and shoulder, pain was relieved by alcohol injection of the sympathetics. This patient died about two months later of a cardiac condition so that we were unable to attach much significance to the result. We feel, however, that in view of the cases reported of recurrent trifacial pain after sensory root section relieved by sympathetic ganglionectomy (Flothow,[9] Mixter and White[10]), that there is a very definite field for alcohol injection in these conditions. Reichert[11] reports such a case treated successfully.

ANGINA PECTORIS

White[12] has done more than anyone else in this disease. The results are most satisfactory. The results in the 3 cases we have to report, have been excellent, although too little time has elapsed since injection for a conclusive report; in each case, however, anginal pain has been relieved. There are several points of interest relative to this treatment. The cardiologist objects to the treatment because he feels that if pain is relieved the patient has lost his warning and death will ensue. Strangely enough, every case we have observed has been entirely relieved of pain but with a warning still present in a sense of suffocation or constriction on over-exertion.

For anginal conditions the upper five dorsal segments on the left side are injected. At times, it may be necessary to inject the right side also.

White reports several cases of painful aneurysm relieved by this method.

BRONCHIAL ASTHMA

We have but 1 case to report, but one of great interest to us because of the marked relief of the asthma and our only mortality to date.

The patient, a man fifty-one years of age, was practically in status asthmaticus, able to breathe sufficiently to sustain life only by a liberal use of adrenalin. Because no other form of treatment was of any avail we were asked to inject his sympathetics. It was necessary to do this with the patient sitting upright due to his extreme dyspnea. The upper three dorsal segments on each side were injected with 5 c.c. of 1 per cent procaine in each needle. The result was quite dramatic. Within a few minutes the asthma began to improve, and within fifteen minutes breathing was almost normal. Feeling that this result might be psychic, we decided not to inject alcohol, but to wait and see how long relief would last. After relief of approximately one hour, asthmatic breathing recurred more severely than before and did not respond as previously to adrenalin, and the patient expired in four hours.

Autopsy disclosed complete collapse of both lungs with bronchioles full of frothy substance. Unfortunately, we were not called until too late to reinject, for we believe that, had alcohol been injected, this patient would be alive and markedly relieved of his asthma.

The explanation of this result is conjecture, but we believe that when the anesthesia of the sympathetics wore off, there occurred a compensatory stimulating effect upon the sympathetics which was unaffected by adrenalin, resulting in complete constriction of the bronchioles.

Stern and Spivacke[13] report a case of bronchial asthma in which unilateral alcohol injection was successful. From Europe come reports of good results following operative removal of the sympathetic ganglia.

TROPHIC ULCERS AND SKIN AFFECTIONS

Under this heading occur a variety of conditions which may be treated by alcohol injection. Many trophic conditions of unknown origin will undoubtedly respond to this treatment, and all conditions of this type for which the LeRiche method of operation is advocated can be more effectively and certainly benefited by alcohol injection.

One of our cases is of especial interest; a woman, fifty-six years of age, with a peculiar skin condition of many years' standing, her skin from the knees down resembling nothing so much as elephant's hide. The color was about the same, it was thick and wrinkled like elephant's skin, and very dry and scaley. A large ulcer on one leg about 3 in. in diameter would not heal, and severe pain in both legs was constantly present. The hospital staff, at a loss as to diagnosis and treatment, prevailed upon us to try an alcohol injection. A diagnostic injection resulted in prompt relief of pain and marked increase in temperature. Alcohol was then injected and to our surprise the skin condition rapidly began to clear up and the ulcer to heal. Within a week the ulcer had healed nearly 50 per cent, the wrinkles were leaving the skin, and where the scales were removed fairly normal skin appeared. The contrast to the uninjected side was very marked and two weeks later this side was injected. Approximately six weeks have elapsed; the ulcer is entirely healed, the skin is improving daily and relief of pain has persisted.

This case leads us to believe that neurotrophic skin lesions which do not respond to the usual treatment might be benefited by alcohol injection.

DIABETIC NEURITIS AND GANGRENE

In 2 cases of painful diabetic neuritis, one associated with gangrene, alcohol injection relieved the pain. In the case in which gangrene was present several toes had been amputated leaving a gangrenous sloughing area which would not heal and was associated with severe pain. Alcohol injection gave immediate relief of pain and the gangrenous area promptly began to granulate and within six weeks was practically healed.

We feel that at least diagnostic injection should be employed in all cases of diabetic gangrene which resist treatment.

MISCELLANEOUS CONDITIONS

Under this heading we are grouping a number of painful conditions which we have not had the opportunity of treating, but which we feel would probably be benefited by alcohol injection.

Migraine. Those cases in which the pain is definitely unilateral are worthy of at least diagnostic injection. Relief of pain should be followed by alcohol injection which would act not only as a therapeutic but as a diagnostic measure which, if effective, could be followed by surgery. Dandy's[14] recent report of cases cured by operation bears this out.

Pain in Malignancies. A diagnostic injection will indicate whether or not alcohol injection should be done. Certainly this would be preferable to the operative treatment which has been advocated.

Painful Amputation Stumps. We feel certain that a good percentage of these cases would respond to this treatment and suitable ones could easily be selected through diagnostic injection.

As a last consideration, we wish to suggest that any pain of vague and unknown origin may be carried over sympathetic channels. It is easy to determine if this is the case merely by a diagnostic injection. It is quite possible that a surprising number of successful results would be obtained.

COMMENTS

Untoward Results. In the main these are the same as those encountered in any procaine or alcohol injection. There are, however, some particularly relevant to alcohol injection, the most important being alcoholic neuritis, probably due to the exposure of somatic nerve roots to alcohol. In the dorsal regions this neuritis is often very severe and most annoying to the patient, usually lasting from two to six weeks, and is best treated by sedatives and heat. In the lumbar injections the neuritis occurs in only about half the cases and is usually not as severe.

In the dorsal region there is danger of penetrating the pleura and injecting alcohol intrapleurally. This has occurred to us once and the resulting pleuritic pain was so intense as to put the patient into shock,

TABLE I

DIAGNOSTIC INJECTIONS

No.	Date	Patient	Sex	Age	Diagnosis	Symptoms	Injection–Level	Temperature Increase	Relief–Pain	Novocaine	Results and Comments
1	9–30–30	I. I.	M	45	Buerger's disease	Pain—gangrene following amputation toe	Labat L. lumbar	7.5°C.	Yes	20 c.c.	Ganglionectomy resulted in relief of pain and healing of gangrene
2	10–10–30	H. R.	M	35	Buerger's disease	Pain gangrene of big toe	Labat Rt. lumbar	0	0	20 c.c.	Increase of temperature to just below the knee, amputation
3	10–12–30	O. A.	F	20	Traumatic Raynaud's disease	Cold, blue, painful arm, following injury to hand	Rt. D. 1–2 Labat	18°F.	Yes	15 c.c.	Complete relief of pain and warming of hand. Ganglionectomy resulted in cure
4	12– 2–30	L. C.	M	39	Buerger's disease	Pain—loss of both legs and several fingers of each hand	Labat Lt. D. 1–2	3.5°C.	Yes	15 c.c.	Ganglionectomy relieved pain and healed gangrene
5	12–10–30	P. L.	M	21	Polyarthritis. Osteitis fibrosa cystica	Pain—ankylosis of all joints both legs	Lt. L. 1–2–3–4	not measured	0	20 c.c.	No relief of pain. Had a convulsion on table. Hopeless case
6	2– 5–31	W. M.	F	40	Polyarthritis	Marked arthritic pain. Cold clammy extremities	Lt. L. 1–2–3–4	12.3°F.	Yes	20 c.c.	Arthritic pain relieved and joint function increased markedly by ganglionectomy
7	3–26–31	R. H.	F	38	Neuritis of leg	Pain in tibia, unknown origin	Rt. L. 1–2–3–4	0	0	20 c.c.	Needles too short in very fat person. Sympathetics missed. Refused reinjection
8	5–22–31	J. V.	M	51	Bronchial asthma	Very severe. Adrenalin gave slight relief	Bilat. D. 1–2–3	not measured	—	30 c.c.	See text. Relief of asthma followed by death as anesthesia wore off
9	6–17–31	M. B.	M	67	Traumatic neuritis	Pain in foot at night for 14 years since ran nail into foot	Lt. L. 1–2–3–4	marked	No	20 c.c.	Marked increase in temperature, but no relief of pain
10	7–22–31	C. W.	F	55	Cancer of breast with brachial neuritis	Intense pain in arm and shoulder	Lt. D. 1–2–3	not measured	No	15 c.c.	No relief of pain. Marked temperature increase
11	7–22–31	C. M.	M	34	Cervical spondylitis	Severe pain in neck and back	Bilateral D. 1–2–3	not measured	No	30 c.c.	Marked increase in temperature. No relief of pain
12	7–22–31	M. M.	F	60	Polyarthritis	Severe pain in joints and neck	Rt. D. 1–2–3	not measured	No	15 c.c.	No relief of pain, therefore not suitable
13	8– 2–31	W. L.	M	42	Encephalitis lethargica	Pain and spasticity of arm	Rt. D. 1–2–3	5°C.	0	15 c.c.	No relief of pain or decrease in spasticity
14	8–13–31	W. C.	F	55	Indeterminate	Pain, twitching of face, numbness of face	Lt. D. 1–2–3	marked	No	15 c.c.	Peculiar condition felt to be psychic. Not sympathetic in origin
15	8–22–31	A. B.	M	63	Spondylitis	Pain in back and hip, ankylosis of hip	Rt. L. 1–2–3–4	0	No	20 c.c.	Unsuccessful injection, to be repeated
16	9–18–31	E. S.	M	82	Spinal cord sclerosis	Severe pain in both legs, atrophy	Lt. L. 1–2–3–4	4°C.	0	20 c.c.	Fair vascular result. No relief of pain. Therefore not sympathetic in origin

TABLE II

ALCOHOL INJECTIONS FOR VASCULAR LESIONS

No	Date	Patient	Sex	Age	Diagnosis	Symptoms	Injection Level	Temperature Increase	Relief Pain	Alcohol	Results and Comments
1	2-18-31	D. R.	M	73	Arteriosclerosis	Pain rt. calf, cold foot	Rt. L. 1-2-3-4	+++	Yes	20 c.c.	Relief for 48 hours, then recurrence of pain. See next injection
	2-21-31	D. R.	M	73	Same	Same	Rt. L. 12 1-2-3	+++	Yes	20 c.c.	Complete relief of pain this time. Neuritis ++++
2	3-18-31	A. G.	M	70	Arteriosclerotic gangrene	Pain and gangrene to ankle	Rt. L. 1-2-3-4	?	?	20 c.c.	Injection immediately before amputation below knee. Healed well, no pain. Neuritis o
3	4-10-31	V. J.	M	58	Arteriosclerosis	Pain—trophic changes, absent pulsations	Lt. L. 1-2-3-4	8.5°c.	Yes	20 c.c.	No more pain, foot warm; amputation because of complete ankylosis
	5-20-31	V. J.	M	58	Same	Same	Rt. L. 1-2-3-4	10°c.	Yes	20 c.c.	Permanent relief of pain. Leg warm, of good color. Neuritis +++
4	4-10-31	J. M.	M	55	Arteriosclerosis Post-amputation toe drainage	Pain—draining gang. area. Cold, vessels not palpable	Rt. L. 1-2-3	++	Yes	15 c.c.	Pain gone. Draining area healed. Foot warm. Neuritis +++
5	4-18-31	H. E.	M	53	Arteriosclerosis or Buerger's disease	Pain—cold blue foot—weakness	Rt. L. 1-2-3-4	12°c.	Yes	20 c.c	Pain gone. Foot warm, pink. Neuritis o
	6- 4-31	H. E.	M	53	Same	Same	Lt. L. 12 1-2-3	10°c.	Yes	20 c.c.	Same result. Neuritis +
6	4-22-31	S. M.	M	50	Arteriosclerosis or Buerger's disease	Cold foot. Pain and cramps when walking	Rt. 1-2-3-4	7°c.	Yes	20 c.c.	Complete relief for 5 months. Recurrence, to be reinjected
7	4-28-31	R. H.	M	57	Arteriosclerosis and C.N.S.Lues	Pain and non-healing ulcers	Lt. L. 1-2-3-4	3°c.	Slight	20 c.c.	Somewhat improved condition, but ulcers probably due to lues. Neuritis o
8	5-13-31	H. N.	M	46	Raynaud's with muscular atrophy	Cold, clammy cyanotic painful extremities	Rt. D. 1-2	6.7°c.	Yes	10 c.c.	Permanent warm, pink, painless hand. Neuritis +++
	6- 4-31	H. N.	M	46	Same	Same in feet	Lt. L. 1-2-3	7°c.	Yes	15 c.c.	As above. Neuritis ++. No effect to date noted on atrophy
9	6-17-31	M. B.	M	73	Arteriosclerosis	Unable to walk for pain both legs. Feet always cold	Rt. L. 1-2-3-4	10°c.	Yes	20 c.c.	Immediate relief of pain; foot became warm. Neuritis ++
	6-24-31	M. B.	M	73	Same	Same	Lt. L. 1-2-3-4	8.5°c.	Yes	20 c c.	Same result, able to walk after injection. No pain, feet warm. Neuritis o
10	7-10-31	J. N.	M	68	Arteriosclerotic gangrene	Pain and gangrene of toes	Lt. L. 1-2-3-4	1.9°c.	o	20 c.c.	Amputation disclosed complete obliteration of all vessels
11	7-15-31	T. V.	M	67	Arteriosclerosis with gangrene	Recurrent pain and gangrene after Le Riche operation	Lt. L. 1-2-3-4	5.6°c.	Yes	20 c.c.	Pain gone. Gangrene healed rapidly. Neuritis
12	7-24-31	A B.	M	35	Buerger's disease	Pain, cold foot, ulceration of ankle	Lt. L. 1-2-3-4	6°c.	Yes	20 c.c.	Pain gone, foot warm, ulcers healed. After 2 months recurrence. Operation advised

Table II (*Continued*)

No.	Date	Patient	Sex	Age	Diagnosis	Symptoms	Injection	Temperature Increase	Relief Pain	Alcohol	Results and Comments
13	9- 5-31	H. T.	M	78	Arteriosclerosis with gangrene	Painful cold foot, beginning gangrene toes	Rt. L. 1-2-3-4	5.8°c.	Yes	20 c.c.	Pain gone, toes back to normal within 2 weeks. Neuritis ++
14	9-16-31	O. J.	M	56	Arteriosclerotic gangrene	Pain—gangrene of toe	Lt. L 1-2-3-4	3°c.	Yes	20 c.c.	Gangrene cleaned up. Pain almost entirely gone
15	9-18-31	A. M.	M	58	Arteriosclerosis. Extensive gangrene entire foot	Sever· pain gangrene up to ankle	Rt. L. 1-2-3-4	4°c.	No	20 c.c.	Alcohol injected as preamputation measure. Caused demarcation and edema disappeared. Good blood supply to stump
16	9-30-31	J. M.	M	50	Intermittent claudication	Pain from knee down when walking, cold feet	Rt. L. 1-2-3-4	12°c.	Yes	20 c.c.	Pain gone. Foot warm and dry. Neuritis o

lasting for forty-eight hours. This should rarely occur as procaine should always precede alcohol to determine by means of the cough reflex whether the pleura has been penetrated.

Cases have been reported of paralysis as a result of injecting alcohol intradurally or intramedullarly. This is impossible if the Lundy technique is used; penetration of pleura is also less common.

Sudden collapse with momentary cessation of either respiration or pulse or both has occurred, usually in the dorsal injection. This occurs just as the alcohol is being injected, for there seems to be a terrific shock associated with the injection of alcohol so close to the pleura even though the patient is under general anesthesia. Although only momentary, as a rule, in one case artificial respiration was necessary.

The Horner syndrome of necessity follows the dorsal injection of alcohol.

Duration of Results. This is the only uncertain factor in this type of treatment. We have noted continued vasodilatation present one year following injection; we have also noted its disappearance within one month. The duration seems to depend upon the proximity of the point of the needle to the sympathetic chain at the time of injection. If a direct injection of the trunk and ganglia is made the result should be permanent, but this probably does not occur in a very large percentage of cases. In some cases, even where vasodilatation has apparently disappeared, pain has not recurred. We now have our first case in which reinjection is necessary: an arteriosclerotic reports recurrence of pain and is to come in for reinjection, after relief for a period of five months.

In the cases in which we have performed injection and operation on the same patient on opposite sides, the operated side invariably shows more evidence of vasodilatation after a lapse of two or three months, although relief of pain may be equal. For this reason we advocate operative measures in those cases where maximum blood supply is essential.

Alcobol Injection versus Surgical Removal. We have already indicated our opinion in the preceding paragraph. Injection will never replace surgery. It can always be used as a substitute for surgery when, for any reason, surgery is contraindicated.

In the dorsal region owing to the severity and duration of neuritis following injection, we feel that surgery is preferable,

TABLE III

ALCOHOL INJECTIONS IN ANGINA PECTORIS AND CHRONIC POLYARTHRITIS

No.	Date	Patient	Sex	Age	Diagnosis	Symptoms	Injection	Temperature Increase	Relief Pain	Alcohol	Results and Comments
1	2-20-31	W. M.	F	40	Polyarthritis deformans	Pain, deformity, marked limitation of motion	Rt. L. 1-2-3-4	+	Yes	20 c.c.	Marked relief of pain and increase in joint function. Progressive improvement. After 5 months recurrence of pain
	4- 3-31	W. M.	F	40	Same	Same	Rt. D. 1-2-3	+	Yes	15 c.c.	Relief of pain, increase in function not as marked as on operated side. Both injections caused neuritis +++
2	4- 7-31	A. B.	F	54	Polyarthritis deformans	Pain, deformity, limitation of joint motion	Rt. L. 1-2-3-4	9°c.	Yes	20 c.c.	Complete relief of pain. Marked increase of motion and temperature increase
	4-29-31	A. B.	F	54	Same	Same	Rt. D. 1-2-3	Marked	Yes	15 c.c.	As above, but not as permanent. Temperature increase gone in 3 months
	6-10-31	A. B.	F	54	Same	Same	Lt. L. 1-2-3-4	7.8°c.	Yes	20 c.c.	Pain relief only moderate in this instance. Temperature increase gone in 2½ months
3	6-10-31	L. S.	M	42	Polyarthritis deformans	Pain, deformity, limitation of motion	Rt. D. 1-2-3	+	Yes	15 c.c.	Relief of pain, increase in function. Temperature increase to date. Neuritis ++
	6-18-31	L. S.	M	42	Same	Same	Lt. L. 1-2-3-4	+	Yes	20 c.c.	As above, in both cases to date injection result comparable to surgical removal done on opposite side
4	4-21-31	F. B.	M	67	Angina pectoris coronary sclerosis	Severe pain, shortness of breath, cyanosis	Lt. D. 1 to 5	not measured	Yes	25 c.c.	No anginal pain since injection. Shortness of breath and cyanosis gone. Neuritis ++
5	6-12-31	T. M.	F	..	Angina pectoris	Severe anginal pain, confined to bed 6 months. Very short breath	Lt. D. 1 to 5	not measured	Yes	25 c.c.	No anginal pain. Able to be about. Neuritis +
6	7-29-31	M.	M	..	Angina pectoris	Severe pain on exertion, abdomen, chest and left arm	Lt. D. 1 to 5	not measured	Yes	25 c.c.	No anginal pain. General condition improved. Neuritis +++

and patients who have had both concur in this.

Alcohol injection can, however, be used in a wide variety of conditions in which an operation of the magnitude of ganglionectomy would not be justified.

The injection is not without danger and should not be done by anyone not thor-

TABLE IV

ALCOHOL INJECTIONS—MISCELLANEOUS CONDITIONS

No.	Date	Patient	Sex	Age	Diagnosis	Symptoms	Injection-level	Temperature Increase	Relief Pain	Alcohol	Results and Comments
1	10- 4-30	G. F.	M	30	Multiple sclerosis	Spasticity	Labat: L. lumbar	15°F.	....	15 c.c.	Marked increase in temperature. No effect on spasticity. Neuritis 0
2	12-10-30	M. M.	F	73	Trifacial neuralgia	Severe facial pain in all 3 divisions	Rt. D. 1-2 Labat	not measured	Yes	15 c.c.	Patient lost track of after 6 months, during which time she had no further pain
3	2-12-31	E. W.	F	33	Elephantiasis	Marked enlargement of leg following Kondoleon operation	Lt. L. 1-2-3-4	15°F.	....	20 c.c.	Leg remained warm for 2½ months. No effect on elephantiasis. Neuritis
4	3-14-31	M. S.	F	78	Atypical facial pain, coronary disease	Pain in face, neck, shoulder	Lt. inf. cerv. 1-2 D	not measured	Yes	15 c.c.	Facial pain entirely relieved. Neuritis so severe that could not determine about neck and shoulder pain. Died cardiac death in 2 months
5	3-17-31	L. R.	F	34	Spastic paraplegia. Chronic constipation	Both legs spastic, constipation since birth	Lt. L. 1-2-3-4	not measured	....	20 c.c.	Considerable reduction in spasticity of both legs. Constipation markedly improved. Neuritis ++
	3-24-31	L. R.	F	34	Same	Same	Rt. L. 1-2-3-4	not measured	....	20 c.c.	
6	4-29-31	N. B.	F	50	Diabetic neuritis	Severe pain in rt. leg	Rt. L. 1-2-3-4	++	Yes	20 c.c.	Pain relief to date. Has some pain relieved by sodium bicarbonate
7	6-1-31	G. D.	M	53	Epigastric pain and burning	Pain of unrecognized origin; thought possibly sympathetic	Lt. D. 6-12	not measured	No	35 c.c.	Following novocaine pain in epigastrium relieved. Requested alcohol injection of other side. After both sides injected pain was as severe as before. Neuritis +++
	6-3-31	G. D.	M	53	Same	Same	Rt. D. 6-11	not measured	No	30 c.c.	
8	7-22-31	E. C.	M	74	Diabetic neuritis, gangrene in toe, amputation stump	Severe pain and non-healing amputated toe stump	Lt. L. 1-2-3-4	3°C.	Yes	20 c.c.	Pain gone to date. Gangrene granulated in, healing rapidly, after removal of infected bone
9	8-19-31	H. H.	F	68	Indeterminate	Pain both legs. Thickening and hardening of skin. Large ulcer on leg	Rt. L. 1-2-3-4	++	Yes	20 c.c.	Immediate relief of pain. Ulcer healed completely within 4 weeks. Skin rapidly clearing up.
	8-26-31	H. H.	F	68	Same	Same	Lt. L. 1-2-3-4	None	No	.....	Owing to deformity failed to strike sympathetics
	9- 5-31	H. H.	F	68	Same	Same	Lt. L. 1-2-3-4	++	Yes	20 c.c.	Immediate relief of pain, progressive improvement in skin to date. See text

TABLE IV (*Continued*)

No.	Date	Patient	Sex	Age	Diagnosis	Symptoms	Injection-level	Temperature Increase	Relief Pain	Alcohol	Results and Comments
10	8-24-31	R. L.	F	35	Malignancy of pelvis	Right side, severe pain in hip and leg	Rt. L. 1-2-3-4	not measured	Partial	.....	Patient felt pain was diminished by novocaine. Alcohol injected; only partial relief of pain
11	9-10-31	G. B.	M	47	Pain and atrophy of rt. leg. Unknown origin	See diagnosis	Rt. L. 1-2-3-4	4°C.	Partial	20 c.c.	Patient felt pain relieved by novocaine. Alcohol injected. Too soon to evaluate but probably a failure. Neuritis +++
12	10- 1-31	A. M.	F	24	Indeterminate; neuritis, left leg	Severe pain and burning in rt. leg, following spinal anesthetic. Cold clammy foot	Lt. L. 1-2-3-4	17.5°F.	Yes	20 c.c.	Novocaine warmed foot and relieved pain definitely, so alcohol was injected. Final result (?)
13	10- 2-31	A. J.	F	28	Indeterminate; severe neuritis lt. arm and neck	Severe pain in left arm, neck and ear, following burn on hand 3 years ago	Lt. D. 1-2-3	7°C.	Yes	15 c.c.	Immediate relief of all pain in arm, neck and ear, with novocaine and alcohol same result severe neuritis from alcohol

oughly conversant with the technique, the anatomy, and the dangers.

SUMMARY

The most satisfactory diagnostic procedure for conditions affected by the sympathetic nervous system is the direct injection of procaine at the sympathetic nerves.

By means of these diagnostic injections it is possible to determine accurately whether or not therapeutic measures should be directed toward the sympathetic nervous system.

The two factors most frequently involved are pain and diminished blood supply. Diagnostic injection gives definite information as to relief of pain and what quantitative increase in vascularity may be obtained by therapeutic measures.

Alcohol injection of the sympathetic nerves is an established therapeutic measure. We have used it successfully in arteriosclerotic conditions, Buerger's disease, Raynaud's disease, chronic polyarthritis, trifacial neuralgia, atypical facial pains, angina pectoris, trophic ulcers, neurotrophic skin lesions, diabetic neuritis, and other conditions.

Tables are presented including 64 diagnostic injections, 48 of which were followed by alcohol injection with uniformly good results.

Alcohol injection may be used as a substitute for surgical removal of sympathetic ganglia where the latter is not indicated.

Alcohol injection does not have the permanency of the operation and is therefore not indicated where permanent maximum blood supply is essential except where operation is definitely contraindicated.

Alcohol injection can be used more successfully than the LeRiche operation in all of the conditions for which this operation has been advocated.

Surgery or alcohol injection on the sympathetic nervous system should be preceded by diagnostic procaine injection. This diagnostic injection has taken guess work entirely out of the realm of sympathetic therapeutic measures.

[For References see p. 625.]

SOME THOUGHTS ON THE PROBLEM OF CANCER CONTROL*

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DURING the last quarter of a century, the subject of cancer control has been receiving an ever-increasing amount of attention, and in the last decade, it has taken first place in the discussions of medical societies throughout the world. Interest on the part of the laity has likewise been rapidly increasing, due in a measure, to the widespread propaganda of the numerous societies for the control of cancer, and also to the fact that the mortality of cancer has been steadily rising, especially during the last twenty-five years. A pioneer among these societies is the American Society for the Control of Cancer, and it is gratifying to note that this society in 1927 had little difficulty in raising the million-dollar endowment which it set out to do. Among the many proofs of this growing interest in the problem of cancer may be mentioned the International Lake Mohonk Conference held in 1926, and the International Cancer Conference held in London in 1928 under the auspices of the British Empire Cancer Committee. Both of these were attended by delegates from all over the world, and interesting and valuable papers were presented by the leading specialists in the field of clinical and laboratory cancer research. I had the privilege of being present at both of these conferences.

A careful review of the reports made on these two occasions leaves one in a very pessimistic frame of mind. No new light was thrown upon the fundamental problem, i.e., the etiology of cancer; and practically nothing new was offered in the way of more efficient methods of controlling cancer or of checking its steadily rising mortality.

A most important paper at the Lake Mohonk Conference was that of Ewing on "The Prevention of Cancer." In the opinion of Ewing, prevention plays a prominent rôle in the control of cancer. He stated there were many reasons for the current neglect of the prevention of cancer. Among the obstacles enumerated was, "the general assumption that cancer is the result of hereditary tendencies which express themselves in the so-called spontaneous outbreak of the disease." However, he did not believe that *heredity* could be dismissed from a practical consideration of the origin of cancer; and he approved of the public being advised that, when there is a strong tendency to cancer in the family, the other members take unusual precautions against the disease.

A far more serious obstacle he believed, was the widespread assumption of the parasitic theory of the origin of cancer. He stated:

> If cancer is due to the action of an unknown microscopic, perhaps ultramicroscopic, universal parasite, then effectual prevention must await upon its discovery. At the present day, I have no hesitation in committing myself without reservation against this theory. With most general pathologists, I regard it as incompatible with the known facts about cancer. The assumption of a universal cancer parasite can be held only by those who assume in addition that cancer is a single disease, comparable to syphilis or tuberculosis. This assumption appears to be untenable. Cancer is not a single pathological entity, but a great group of diseases, of very varied origin and course.

And yet, Ewing admits that neoplastic reaction of tissue cells is comparable to

* Read before the Tri-State Medical Association, Memphis, Tenn., February, 1931.

inflammatory reaction, which latter, is due to a microbic cause.

According to Ewing, a rational basis for the prevention of cancer lies in the fact that the major forms of the disease are due to some form of chronic irritation. In closing, he pointed out that early diagnosis alone is not capable of accomplishing the desired reduction in the death rate; that the patient coming with an early diagnosis all too often fails completely of a cure. He states, "The experience of patients accomplishing a cure of early cancer is generally a severe one, while the fate of the failures is passed over in silence. The public knows these facts and therefore any plan of squarely meeting the problem of cancer control must eventually lean heavily upon cancer prevention."

While it may be possible to avoid or prevent a certain amount of chronic irritation, in the great majority of cases the chronic irritation which is supposed to be the exciting cause of the malignant tumor is associated with the occupation or routine activity of the individual, and cannot well be avoided unless one is able or willing to give up work or all physical activities and lead a life of leisure.

RESULTS OF SURGICAL TREATMENT OF CANCER

According to some statistics, it has been found possible to obtain from 30 to 50 per cent five-year recoveries in cancer of the breast by early operation, and 33 per cent five-year recoveries in cancer of the cervix by surgery and also by radiation. However the occasional results reported by a few surgeons of large experience and unusual skill fail to give a true picture of the actual average results following the surgical treatment of cancer. This was recognized by Gibson.[1] His unique report of 583 cases of malignant disease observed on the First Surgical (Cornell) Service of the New York Hospital, from February, 1913 to January 1, 1926, showed that of 437 patients operated upon, 308 had died; only 64 were known to be alive and free from recurrence at the time of the report, and only 13 were alive for a period of five years after operation. According to Gibson: "No sadder report of the disheartening status of cancer surgery has come to our attention. It is however inevitable, dealing only with facts . . . Similar research emanating from other institutions would doubtless furnish surprises. We have been living in a fool's paradise."

One month after the publication of Gibson's paper, the end-results of treatment by operation and radiation in 376 cases of cancer of the mouth, observed at the Massachusetts General and Collis P. Huntington Memorial Hospital during the years 1918, 1919 and 1920, were brought out by Simmons.[2] In brief, only 20 patients were well for a period of three years; 16 of these were treated by surgery, and 4 by radiation. In the surgical cases only was the diagnosis confirmed by microscopical examination. Of 108 cases in which the regional glands were involved at the time the treatment was begun, there were no cures.

Shore,[3] has given us one of the most complete and valuable set of statistics dealing with the operability of cancer. This paper is based upon a study of 1000 cases of cancer observed at St. Luke's Hospital, New York City, from July 1923 to January 1927.

1. 62 or 6.2 per cent refused admission as hopeless.

2. 306 or 30.6 per cent admitted but not regarded as suitable for operative surgery.

3. 632, or 63.2 per cent admitted for treatment.

Of the 320 patients operated upon 26 or 8.11 per cent died. Only 29.4 per cent left the hospital with an assurance that the malignant process had been removed. Shore observes: "Therse are indeed discouraging figures in the surgical treatment of cancer."

The conclusion is that 68 per cent of the 1000 patients were found inoperable when they reached the hospital. Many more of the remaining group died of recurrence.

Wood, in a later paper, based on a study of 748 cases of cancer, found only 135, or 21.8 per cent mechanically removable at time of observation.

These statistics represent the results obtained by a few men of unusual skill and experience. The average results throughout the country give an even more gloomy picture, and it is upon the average results that the opinion of the laity is based. Realizing this, the reason for their pessimism becomes apparent. In spite of all the recent propaganda urging the patient to consult a doctor on the slightest suspicion of a cancer, it has been found that in many cases, even of the breast or tongue, where early recognition would seem certain, the patient himself has not been aware of the existence of a cancer until it had involved the regional nodes.

The most discouraging fact in connection with cancer control is, that more than one-half of all cases of cancer occur within the abdominal cavity; and in this large group of cases, the disease has usually progressed too far before the diagnosis is made to justify much hope of curing it by any known method. While a considerable number of cases of cancer of the stomach have been cured by surgery performed by men who have devoted years to the perfection of their technique in this field, the fact remains that the vast majority of patients with cancer of the stomach die of the disease in spite of any treatment they are able to obtain. The best statistics do not show more than 2½ per cent of five-year cures in cancer of the stomach. The same is true of cancer of the liver and intestines. Therefore, we see that practically one-half of all patients with intra-abdominal cancer are destined to die of the disease in spite of any improvement in diagnosis and treatment.

The most discouraging feature about cancer, is its steadily rising mortality, which, according to Hoffman, is apparently universal. In 1885 the recorded cancer death-rate of Scotland was only 32 per 100,000, increasing to 41 by 1865 and to 48 by 1875. In other words, during the twenty years intervening, the cancer deaths increased exactly 50 per cent. The rate increased to 57 by 1885 and during the next ten years to 72, by 1895. The relative rise in the rate during these twenty years was, therefore, again exactly 50 per cent. By 1905 the rate had increased to 91 and by 1915 it had reached 111, or an actual increase in twenty years of about 39 per 100,000, or 54 per cent of the rate at the beginning of the next period. During the five years following, the rate increased to 119 and during the last three years, ending with 1923, to 130. This, in other words, is the highest rate ever reported in the history of Scotland. Thus, in forty years, or between 1884 and 1923, the cancer rate has more than doubled, or, precisely, has increased from 56 to 130 per 100,000, equivalent to about 132 per cent. Hoffman has placed the increase in the cancer death-rate at about 100 per cent during the last forty years.

I would direct attention to some equally interesting statistics for the province of Ontario. According to the last registration report, the cancer rate of that province in 1914 was 69.6 per 100,000, which by 1918 had increased to 75.5 and by 1923 to 90. Cancer of the buccal cavity underwent slight changes, but there was a material increase in cancer of the stomach, from 22.8 in 1914 to 31.0 in 1923. Still more suggestive is the increase in cancer of the peritoneum and the intestines: from 9.8 in 1914 to 14.1 in 1923. Cancer of the female generative organs increased from 5.2 in 1914 to a rate of 9.4 in 1923. Cancer of the breast, which is certainly an easily diagnosed portion of the body, increased from 4.5 in 1914 to 9.2 in 1923. Cancer of the skin also increased, from 0.99 in 1914 to 2.7 in 1923. Other cancers, including unspecified ones, have remained practically stationary, the rate in 1914 having been 20.5, while in 1923 the rate was 19.2. It is pointed out in the report that the total number of deaths from cancer in the

Province reached 2724 for 1923, or probably nearly 3000 for 1924. During the last decade the rate has increased at the rate of 20.4 per 100,000 of population. Comparing the mean rate of the last decade with that of the preceding decade, there has been a relative increase in the cancer death-rate of 31 per cent.

A study of the mortality statistics of the United States shows practically the same steady and marked increase in the death-rate from cancer in every state. Cancer has now reached second place among the causes of death and is rapidly approaching first place.

Before the Senatorial Committee in 1930 Dr. Joseph C. Bloodgood said:

> Twenty years ago cancer occupied eighth place among the principal causes of death in this country. Its ascendency to second place presents a vivid picture of the steadily increasing devastation caused by the disease. Cancer now is responsible for 10 per cent of the deaths from all causes in this country.
>
> This increase has taken place in spite of the educational campaign of the American Society for the Control of Cancer during the seventeen years of its existence, in spite of cancer weeks and cancer days, in spite of much publicity through the daily press and the magazines, in spite of an increasing amount of radium available for the treatment of cancer, in spite of the improvement in and the standardization of hospitals in this country, in spite of the immense improvement in medical teaching, in spite of the fact that a large number of people are enlightened and come earlier.
>
> Therefore, if more than 100,000 die annually, there must be from 300,000 to 500,000 cases of cancer living in any one year, who have either received treatment or not. I am inclined to think that more than 50 per cent of them have reached the hopeless stage, notwithstanding treatment by surgery or radiation. I think that a consensus of most of the writers on this subject is that there are about 360,000 cases existent in the United States today.
>
> All cancer students agree that the cause of cancer is undiscovered, except that chronic irritation can produce cancer in experimental animals and in the human being, but we do not know why it is so produced. All cancer students agree that at present there is no specific cure for cancer outside of its removal by surgery or its treatment by radiation and we have demonstrated that the results of such treatment in late cases are less than 10 per cent and in the earliest cases have reached about 50 per cent; but in a large per cent of all cancers—at least 30—the disease is hopeless from the onset, in spite of surgery or radiation.
>
> We have sufficient evidence to know that cancer will never be eradicated completely as the cause of death until we have discovered the cause of the disease and the preventative and curative treatment. No matter how enlightened the individual is or how early he seeks treatment after the first warning, certain types of cancer are inaccessible, or become hopeless before the first symptom is noticed. Or, the cancer cell is of such a type that it disseminates at once before surgical removal is possible, and it is not radiosensitive which means that radiation does not kill the cancer cells. Just as we need more education, continuous, systematic and organized, we need more and better organized research.

Before the same committee Dr. James Ewing stated: "My own experience and general judgment as a pathologist relates to the proportion of cases of cancer that are now cured in the average good hospital service in this country, and I find that it is not more than ten per cent."

Moynihan, in the Hastings Lecture (*Lancet*, January 2, 1927) discusses cancer and how to fight it. He believes that the oft-repeated attempt to explain away the steadily rising mortality of cancer on the ground that (1) people live to a greater age now than formerly, and (2) that in earlier times there were more errors in diagnosis, has no foundation in fact. While he emphasizes the importance of the early diagnosis and the early surgical removal of cancer, he does not rest content with this alone. He would concentrate our attention upon two points, laying greater stress upon the second: (1) to make the very utmost of our present methods which are applicable to all cases of accessible growths; and (2) to undertake research so that we may discover the cause or causes of cancer and so be enabled to do something for the pre-

vention of the disease or for its cure by methods other than surgery.

The present methods of cancer control are based entirely on the theory that cancer is of intrinsic origin, that is, due to something inherent in the cell itself, and not to the influence of some extrinsic agent such as a microorganism.

The great majority of pathologists today are firm believers in the intrinsic origin of cancer. They dominate not only the laboratory field of cancer research, but the clinical field as well; and they are on record as deprecating the time and money spent in further research along the lines of a microparasitic origin of malignant tumors. As a matter of fact, very little time and money have been spent in research along the latter lines in recent years, for the simple reason that the directors of cancer research laboratories are so strongly opposed to the extrinsic theory that young men entering the field of cancer research hesitate to engage in a study so strongly condemned. Futhermore there has been a steady attempt to regard the question as closed and definitely settled. No field of medical research has ever been so completely under the influence of authority as the field of cancer research.

Tradition and authority are of great value in medicine as in any other science, but there comes a time when the investigator must forget the existence of both and concentrate his whole thought and energy upon the discovery of new facts; he must apply the principles of logic, both inductive and deductive, and endeavor to arrive at some definite conclusions. When these have been reached and they seem to be founded on indisputable facts, he should be willing to stand by them, irrespective of whether or not they conflict with opinions handed down by tradition and supported by authority.

Ewing's assumption that "If cancer is really caused by an unknown parasite, then prevention is not to be considered" does not seem to rest upon any logical basis. If we have found out by experience that cancer is often the result of chronic irritation or of local trauma, it seems both logical and natural that we should take all possible steps to avoid these predisposing factors, quite irrespective of whether we do or do not know the real or underlying cause of the disease. One might as well argue that we should not have made any attempt to improve the hygienic conditions in the prevention of tuberculosis before the germ was discovered, although it was well known that persons living under very bad hygienic conditions were more likely to develop the disease. All the known methods of preventing cancer and all the improved methods of treating it should be carried out while we are making most strenuous efforts to discover the cause of the disease. I believe it is a mistake to discourage investigation along the lines that offer the most hope of ever gaining control of the disease, and decidedly discouraging is the statement from a recent leaflet of the American Society for the Control of Cancer:

> If cancer is really due to an unknown parasite, then prevention is not to be considered; and we are not much better off than the ancient Egyptians, since we may at any time be stung to death by the hobgloblin parasite in its own mysterious way. The distressing difference in favor of the Egyptians is that they were beautifully embalmed like Tut-Ankahmen, and we are not.

Those of us who still firmly believe that cancer is due to some extrinsic cause will not be seriously disturbed by the substitution of ridicule for argument; but such statements coming from one of the most brilliant workers in the field of cancer research, cannot fail to discourage investigations along these lines.

In the *Annals of Internal Medicine*, May, 1930 (reprinted in the *Journal American Medicine Association*, June 7, 1930) an editorial writer (Warthin) on "Cancer Cures" makes a most uncompromising attack upon the germ theory of cancer. While in his opinion there was some reason for entertaining the idea that

cancer might be of an infectious nature in the latter part of the last century, he states that by the end of the first decade of the present century experimental work on animals together with the fuller bacteriologic and pathologic studies of neoplasms "had convinced us that cancer is not an infectious disease; that no specific agent exists for the production of cancer." He claims:

> Mistaken conceptions regarding certain infectious growths in the lower animals, particularly the so-called Rous chicken sarcoma, have been in part responsible for the persistence of such views. By many workers the chicken sarcoma is regarded as an infectious granuloma and not comparable to the true neoplasms in man. Furthermore, there exists today absolutely no proof that infection plays any specific part in the production of neoplasms; and when once the public mind has become cognizant of the fact that cancer has no infectious etiology, much will have been accomplished to prevent patients from falling victims to cancer cures.

While this writer brushes aside as of little importance the vast amount of research work done during the past twenty years on filterable fowl tumors on the assumption that these tumors are not infectious granulomata but are comparable to true neoplasms in man, it is curious that Murray, the Director of the Imperial Cancer Research Fund, regards these tumors as by far the most promising field in cancer research today. In his opinion these tumors now have been proved to be true neoplasms comparable to malignant tumors in mammals. A large part of the ninth scientific report of the Imperial Cancer Research Fund is devoted to further studies of the filterable fowl tumors.

Furthermore, the etiology of the filterable fowl tumors still occupies the first place in the cancer research work of the Rockefeller Institute, carried on by Murphy and his co-workers.

If it is true that these tumors are probably due to a living agent, it might be wiser to be a little less positive than was the writer of the editorial referred to, that cancer in man cannot possibly be due to a similar living agent.

That the filterable fowl tumors are true malignant tumors and not infectious granulomata as claimed by the editorial writer in the *Annals of Internal Medicine*, is proved by the fact (as shown by Murray) that metastases in other organs, e.g., the liver or lungs, are always made up of cells corresponding to the type of cell found in the primary tumor, exactly as occurs in human cancer.

These tumors are now recognized in all the important Cancer Research laboratories of the world as true tumors and not infectious granulomata.

Let us study for a moment the main points in favor of the microbic origin of cancer:

1. The close resemblance of certain cases of cancer to other diseases of known microbic origin, for example, tuberculosis and syphilis.

2. The frequency with which there is both a local and general rise in temperature in cases of malignant tumor. This is especially true of endothelioma of bone and other types of cancer with rapid generalization, in which the general temperature often rises to 103–104°F.

3. The difficulty in distinguishing tuberculosis from sarcoma and chronic osteomyelitis, or osteitis from endothelial myeloma, either by the clinical evidence or, in some cases, even by a study of the histological appearance. In a few cases it is impossible for the most experienced pathologist to differentiate these conditions.

4. Single antecedent trauma is now generally conceded to be an important causative factor in malignant tumors, especially bone sarcoma. No satisfactory explanation of the influence of trauma has ever been offered except on the assumption that the malignant tumor was due to some microorganism. Granting this, the explanation is easy. We have an exact parallel in cases of osteomyelitis as well as tuberculosis of bone, following local trauma.

5. The antagonistic action of the streptococcus of erysipelas, and the toxins of erysipelas and Baccillus prodigiosus, upon various types of malignant tumors, is difficult to explain except on the theory that malignant tumors themselves are caused by some microorganism.

6. The remarkable variation in the geographical distribution of cancer. The most recent illustration of this may be found in the Westmoreland County Survey.[12]

Ewing and many other pathologists have stated that cancer is not a single disease, but a group comprising a large number of separate and distinct diseases, and in order to assume a microbic cause one must assume a large number of different microorganisms.

This assumption is by no means necessary. Sir Charles Ballance, at a meeting of the American Surgical Association twenty-five years ago, stated that he was firmly convinced not only that malignant tumors were due to some microorganism, but further that it would probably be shown that the same microorganism, under different conditions, gave rise to the different types of cancer.

Erwin Smith proved this to be true as regards malignant tumors in plants. By making superficial injections he produced epithelioma; by deep injections, a sarcoma; and by injecting a latent bud he produced a teratoma.

In a series of experiments recently carried out at the Laboratory of the Hospital for the Ruptured and Crippled by Dr. Richard F. Berg, using the dried virus of the filterable endothelioma fowl tumors, kindly furnished me by W. E. Gye, of London, found it possible to produce 5 different varieties of bone sarcoma, by injecting the virus into the marrow of the tibia of very young Rhode Island red chicks, one to two weeks old. These tumors correspond very closely to the several types of bone sarcoma in man.

The filterable fowl tumors, which are now almost universally accepted as true neoplasms, comparable to malignant tumors in man, are believed, by the majority of investigators, to be due to some microorganism or living agent, although up to the present time it has never been isolated.

Conner, of San Francisco, had previously been able to produce endothelial myeloma by the same dried virus. Our five varieties corresponded exactly with the different varieties of bone sarcoma in man.

During the past year further experiments with this virus and with the tumors produced by it have been carried on by Dr. J. E. Sullivan (Dr. Berg's successor to the Gibney Memorial Fellowship, the Hospital for Ruptured and Crippled) and myself. These experiments I believe offer convincing evidence that these tumors are caused by a living agent. The full results will be published in the near future.

It would be possible to explain the different types of malignant disease in two ways:

1. The microorganism attacks a particular cell which, by reason of some specific factor, e.g., local trauma, chronic irritation or some chemical changes in the body fluids, offers a lowered resistance to the microorganism. By reason of this lowered resistance, the organism gains a foothold or finds a favorable soil for development, and thus begins the early stages of a malignant tumor. The microorganism enters the cell itself, becoming an intracellular parasite which by its constant irritation, stimulates the cell to growth and division. This growth is naturally made up of the multiplication of the cell in which the organism first enters, and this very naturally accounts for the remarkable fact that the metastases of a malignant tumor always correspond morphologically with the type of cell of the primary tumor. This has been one of the main arguments of the pathologists against the parasitic theory, but if we assume that we are dealing with an intracellular parasite, then the difficulty is at once overcome.

2. We may suppose that instead of a single strain there may be a number of strains of this unknown microorganism, closely related and, perhaps, indistinguishable under the microscope or by variations in the manner of growth upon culture media, which strains may produce different types of tumors.

Personally I am inclined to believe in a single microorganism possessing different degrees of virulence, which, having gained access to the blood of a healthy individual, remains latent until the natural resistance of the cells has been lowered in some local area by some special factor, e.g., local trauma, chronic irritation or chemical changes in the body fluids, furnishing the organism with a favorable soil for its development.

In addition, there may be some inherent or inherited susceptibility of the cells of a certain individual, or an unusual lack of resistance that may account for the varying degrees of what we call malignancy, without assuming a great change in the virulence of the microorganism.

What determines the type of the tumor may not be the germ but the soil or some inherent factor in the cell itself which attracts the organism to one type of cell in one individual and to another type in another individual. If the Rous sarcoma virus is injected into the blood of a susceptible fowl, no tumor develops. Not until an injection is made into areas in which connective tissue is present and a connective tissue reaction is produced, is it possible to produce new tumors. The Rous sarcoma is a spindle cell sarcoma.

The theory that cancer is due to some infectious or microbic cause received great impetus and support by the discovery of the filterable fowl tumors by Fujinami of Japan and Peyton Rous of the Rockefeller Institute (1910). It was found that these tumors could not only be produced by living transplants, as had been done in rat sarcoma and mouse carcinoma, but, by a cell-free filtrate. Then for a time an attempt was made to explain these tumors as not being true neoplasms but similar to the so-called infectious granulomata. The virus itself has been studied in many laboratories, and various opinions have been held as to its true nature, one group of investigators, the larger, believing it to be a living agent, and another, a so-called enzyme or a chemical agent. The remarkable work of Gye of London in July, 1925, gave a new impetus to the opinion that cancer is due to a microbic cause, and the London *Lancet* gave his address a four-page editorial and intimated that at last the age-long problem of the cause of cancer had been solved.

Gye, basing his views upon a long and elaborate study of the Rous sarcoma, evolved the theory that cancer (malignant tumors in general) is not due to a single cause but to a dual cause. He believed: (1) that the Rous sarcoma tumor was due to a "living agent" which had not yet been isolated; (2) that this living agent alone could not produce malignant tumors, but required some other agent, which he called a *specific factor*, to produce the disease. His experiments seemed to prove the truth of this brilliant theory. He first rendered inert the active virus of the Rous sarcoma by chloroform or some antiseptic, until it could no longer produce any tumors on inoculation. Then by adding some one of several substances to this inert material, it once more became active and produced the Rous tumor on inoculation. This discovery was regarded at first as epoch-making, and seemed to go far toward solving the age-long problem of the cause of cancer; but, unfortunately, no one was able to repeat these experiments; and the most rational explanation would seem to be that the living virus was not entirely destroyed by the chloroform and that the addition of the *specific factor* simply helped to revive the inactive but not entirely dead virus, and rendered it capable of producing typical tumors.

Gye's[4] theory differs but little from my own in a general way. According to my theory[5] there is a dual cause: (1) the living

agent which may be widely disseminated and is often present in the tissues or in the circulation of many individuals but doing no harm; (2) Some other factor, not so highly specific as Gye's, for example, local trauma, chronic irritation, or some chemical change in the body fluid, that damages some local area thereby causing a tissue reaction or a favorable soil for the organism to gain a foothold; and thus the beginning of a malignant tumor.

Hereditary susceptibility is doubtless an additional factor in many cases. It means that certain individuals have inherited tissues or cells that are less resistant to the cancer organism when exposed to it.

My theory does not depend on the success or failure of a series of laboratory experiments such as Gye described. It has numerous clinical facts to support it. Furthermore, recent laboratory investigations furnish evidence in its favor.

Cramer, in the ninth Scientific Report on the Investigations of the Imperial Cancer Research Fund, reports a series of experiments in which he was able to transmit three strains of mouse sarcoma and Rous sarcoma without the presence of living cells. While numerous experiments along these lines have been made in the past, all seemed to prove that the transmission of mammalian neoplasm depended on living cells. Three different tumor strains were used by Cramer in his experiments: (1) rat sarcoma (2) different strains of mouse sarcoma, and (3) mouse carcinoma. The mouse carcinoma gave negative results.

These experiments would seem to place neoplasms in mammals measurably nearer, if not in the same class as the filterable sarcomas of Rous and Murray.

Among the clinical facts that furnish evidence in favor of the theory that malignant tumors in man are due to a microorganism or an infectious virus of some kind, the most important and convincing is the case reported by Lecène and Lacassagne.[6] Here a medical student, while attempting to aspirate a collection of lymph from under the scar of an amputated breast (removed for cancer eleven days previously) pricked the palm of his left hand, at the same time discharging a small quantity of the liquid that was in the syringe. Three-quarters of an hour later the wound was cauterized with a galvanic current. The accident was forgotten until two years later when he began to have diffuse pain in the same hand. Shortly after he noticed a bosselated swelling at the exact site of the previous puncture. One month later an enlarged gland, the size of a nut, appeared in the axilla. This gland was removed and was thought to be tuberculous, although no evidence of the disease was found. Four months later the skin of the hand became invaded and ulcerated. One month later the tumor was excised under ether anesthesia. It recurred promptly and showed unmistakable evidence of being a malignant tumor. About five weeks later, four nodules appeared in the skin of the forearm; and a week later, disarticulation of the arm at the shoulder was performed. A careful histological examination of all the nodules and tissue removed at the various operations showed the same general structure. A diagnosis of spindle cell sarcoma was made.

Lecène and Lacassagne, after discussing various hypotheses expressed their conviction that:

> The transmission of cancer is accomplished by the inoculation of a virus analogous to the filterable virus admitted by Gye, possibly of an infectious principle analogous to that admitted by Carrel. The change in the histology of the type of the tumor is entirely "reconcilable" with this hypothesis.*

In my own early experience I recall a case that points to the possibility of a lymphosarcoma of the axilla having been caused by a microorganism introduced in the finger by the prick of a thorn. A young

* In our experimental production of bone sarcoma in young chickens, at the laboratory of the Hospital for Ruptured and Crippled, Dr. Richard Berg, by injections of the virus of the endothelial fowl sarcoma was able to produce not only several varieties of sarcoma but, in two cases, typical carcinoma.

woman while picking roses, stuck a thorn into her index finger. Two or three weeks later, a swelling developed in the axilla of the same side, and grew rapidly. I removed it by operation and it proved to be a round cell sarcoma. It recurred promptly and finally caused death by general dissemination. I was greatly impressed at the time of a causal relationship between the wound of the finger and the rapid development of the malignant tumor in an axillary gland of the same side. I still believe that a microorganism was introduced into the circulation by the thorn-wound of the finger and that it found in the gland of the axilla a favorable soil for development, causing the malignant tumor that proved fatal in less than a year.

Relative to the Part Played by Trauma. If we inject the active virus of a cell-free filtrate of the Rous sarcoma intravenously into an animal, no tumor develops. On the other hand, Pentimalli[7] has shown that in a fowl bearing filterable sarcoma No. 1, a new tumor can be started by applying trauma to some other part of its body. The only explanation of this that can be offered is, that the trauma produced the connective tissue reaction that is essential to the development of a Rous tumor.

This experiment has a very important bearing on the question of trauma as a causative factor in the development of malignant tumors in man. Also it seems to furnish a striking analogy to the experimental production of osteomyelitis in rabbits. It has been found that a sharp blow to the tibia of a rabbit produces nothing unusual; but if the animal is first inoculated intravenously with a small quantity of Staphylococcus aureus and then struck a blow on the tibia, an acute osteomyelitis will develop. In other words, the local trauma furnishes the tissue reaction or soil favorable for the localization of the germ which is present in the circulation but quite harmless until the local resistance of the tissues has been lowered. If this theory is correct, then we have for the first time a rational and scientific explanation of how trauma can be a causative factor in the development of malignant tumors.

One of the most recent and strongest arguments in favor of the microbic origin of malignant tumors, is found in the monograph of Prof. Léon Bouveret[8] of Lyon, France, In addition to the clinical evidence in favor of the theory, he believes the most important and most conclusive evidence is found in the inhibitory, and often curative, influence of the streptococcus of erysipelas upon malignant tumors. This influence, recognized as far back as the time of Hippocrates and reported in a gradually increasing number of cases, is indisputable. Bouveret reports some personal cases and refers to others in French literature. He has carefully examined the various explanations of the curative effect of erysipelas on cancer, and can find only one that is at all satisfactory, i.e., that malignant tumors, themselves, are caused by some strain of streptococcus, and that the curative action of the erysipelas is brought about by antibodies in the blood. This is the explanation that I offered in my first paper 1893 in which I reported the results of treatment of 12 cases of inoperable cancer with the living cultures of the streptococcus of erysipelas; and I have continued to hold this opinion ever since. I would not go so far as Bouveret in assuming that the causative agent of cancer is a strain of streptococcus of erysipelas. I do not believe it essential to assume such a high degree of specificity that it could be acted on only by the same, or a strain of the same organism. We know that the tumors of leprosy have been caused to disappear by a combination of the toxins (vaccines) of the tubercle bacillus and the Bacillus pyocyaneus, quite as rapidly and completely as by a combination of the B. leprosy and B. pyocyaneus. The fact that we have been able to cause in a considerable number of cases the total disappearance of malignant tumors of practically all types by the mixed toxins of erysipelas and B. prodigiosus (which I began using in 1892) proves that heterogenous toxins may have the same

curative action as the autogenous. I would leave open the question as to the type of organisms that is the causative agent of malignant tumors; but that such tumors are caused by some living extrinsic agent, I believe, with Bouveret, that this action of the erysipelas furnishes the strongest kind of proof. Bouveret states:

"It is a fact generally admitted without dispute, that an acute febrile malady may cause a malignant tumor to regress." He believes it still remains to interpret this fact under the influence of diverse febrile conditions: a sarcoma, a lymphoma, and an epithelioma undergo a certain reduction in size. But, he adds, there is found a fairly pronounced regression that may occasionally go on to complete disappearance of the tumor. One finds this among the published cases only in those associated with an attack of erysipelas. To this general rule, Bouveret has found in his researches only two exceptions. Riffel[9] has seen cases of cancer of the face and of the stomach, whose evolution was suspended by an attack of intercurrent variola. In one case of Kutzner,[10] an attack of pneumonia caused a regression of a cancerous neoplasm. I have had a case of sarcoma of the tonsil nearly disappear after an attack of pneumonia, but it promptly recurred and proved fatal within a few months. There can be no doubt that the influence of erysipelas upon malignant tumors is much more powerful than any other febrile disease. Bouveret states that he has never noted regression following attacks of "la grippe," typhoid fever, eruption fevers, or septicemia. On careful inquiry in two hospitals for incurable cancer, he found no history of a case of cancer showing notable regression following intercurrent febrile disease such as bronchopneumonia, although there were many examples of such association. Bouveret asks: "Why is the influence of the erysipelas so preponderating?" and he adds, "Is it not permitted to presume that there exists some relation between the erysipelas and the cancer?" In other words, may not malignant tumors themselves be caused by some microorganism, possibly by some strain of streptococcus closely allied to the streptococcus of erysipelas?

Bouveret has given the most careful study and discussion of the explanation of the regressive action of erysipelas upon malignant tumors that has ever been published, and a brief review of this argument should prove of interest. He points out that the explanation that has usually been accepted in the past, and the one that for the most part obtains today, is, that of "hyperthermie" or high temperature. This is the explanation that is offered by Willy Meyer in his recently published book on "Cancer."[13] It is one, however, which, at the time of my early experiments with inoculations of living cultures of erysipelas (1891–1892) I stated could not satisfactorily account for the inhibitory and often curative influence of an attack of erysipelas. The theory that I offered as the only one that could rationally explain this action, was: that malignant tumors themselves are due to some microorganism; and that the antagonistic and inhibitive action of the erysipelas streptococcus is due to certain changes in the blood serum brought about by antibodies which destroy the causative agent of the tumor, and that later, the degenerating tumor is either absorbed or undergoes extensive necrosis requiring incision and evacuation.

Bouveret, in agreeing with my early explanation, offers the argument that, if fever were the true, efficient and only cause of the marked regression of a malignant neoplasm, such regression would always follow an attack of erysipelas, especially in cases associated with a severe attack of ersyipelas with unusually high temperature. As a matter of fact, he states that the very severe attack with high fever does not generally produce the most marked regression.

Again, if high temperature alone were the sole cause, then all febrile diseases associated with high temperature and also with malignant tumors, should show

marked regression. Such we know is not the case. Clinical observation shows, without any question, that the greatest number of cases of regression are associated with erysipelas alone.

I do not agree with Bouveret that the most marked regressions are not associated with the more severe attacks of erysipelas. In my experience I have found that, as a general rule, the more severe the attack of erysipelas, the more marked the regression, but this I would by no means attribute to the higher temperature but to the production of antibodies in greater numbers and of higher antagonistic power. Bouveret states:

> Erysipelas has the power of curing rebellious ulcers, lupus or syphilis. Is fever the exclusive agent of these cures? These old ulcers have not healed because they are the seat of a chronic, tenacious, microbic infection nor can it be the fever of the erysipelas that destroys the microbes of these infectious processes.

Borrel has made a study of the effect of fever upon pathogenic microbes: "The fever has a salutary significance, not because the microbes suffer from the elevation of temperature, but because it seems to destroy the processes of defense."

Duclaux[11] gives, after the work of Sternberg, the temperature necessary to destroy pathogenic bacteria. The staphylococcus dies at a temperature of 62°C. and the streptococcus at 54°C. Hence it is readily seen that the temperature of erysipelas, 39° to 40°C., falls far short of the degree necessary to destroy the microorganisms present in these old ulcers. Bouveret concludes that it is not the fever then that cures these rebellious, old, tuberculous or syphilitic ulcers, but some other mode of action. Why cannot this other mode of action, or agent, accordingly, cause a regression of malignant tumors?

If, then, the fever is not the essential cause of the regression, what is the cause? It must be the infectious state or infectious nature of the erysipelas. How does it act? The organism tries to defend itself against the erysipelas infection. The general defense of the organism against an attack of erysipelas is assured, not only by the phagocytes, but also by the antibodies in the blood, antibodies which reduce or suppress the virulence of the streptococcus and neutralize the toxins it has produced.

Although Borrel in 1908 found that a transplanted rat sarcoma when subjected to a temperature of 37°C. for twenty-four hours lost its virulence, Haaland (Contamin Le Cancer Experimental, p. 130) studied the effect of heat upon mixed tumors, epithelioma and sarcoma, and found that a temperature of 44°C. for twenty-five minutes did not destroy the virulence of the sarcomatous cells. Fujinami and Inamaoto (Ménétrier: Le Cancer, p. 164) held that although it destroyed the sarcoma cells of filterable fowl tumors, it did not destroy the virulence of a fragment of tumor subjected to a temperature 40° to 50°C. since it was found possible to inoculate successfully other fowls with the heated fragment. Doyen (Congress de Chirurgie, 1908) concluded from a series of researches, that the cancer cell was destroyed by a temperature of 56° to 58°C. Of course, such a temperature is vastly higher than that which occurs in an attack of erysipelas.

Once the truth of the parasitic theory of cancer is proven then a vast new field of scientific research is opened up, and this field holds out the promise of greater success in controlling the disease. We do not need to look for an immediate serum or vaccine that will effect a cure in a large proportion of malignant tumors that have already developed. Such a cure was looked for immediately on the discovery of the tubercle bacillus, and some of us remember the disappointment that followed Koch's failure to produce such a serum. We must expect the long period of observation and further investigation that followed the discovery of the tubercle bacillus. While no vaccine has been found that has proved of great value in the treatment of tuberculosis, knowledge of the habitat of the

tubercle bacillus and of its probable mode of access in the human body, has enabled us to develop preventative measures of great value, and now the mortality of tuberculosis is scarcely more than one-third of what it was before the germ was discovered. Observation revealed that the organism was found frequently in other animals especially in cows, and that many cases of tuberculosis in children resulted from the use of unpasteurized milk from infected cows. This discovery enabled us to institute preventative measures of great value.

In my own opinion, it would seem most probable that the microorganism supposed to be the cause of malignant tumors is very widely distributed; that practically everyone is exposed to it; that it is much more prevalent in certain countries and localities than in others, and that that is the chief reason why we find the wide variations in the incidence of cancer in different localities some of them in close proximity to one another. This theory is strongly supported by the recent survey of Westmorland County Survey.

In this connection, the investigation into the incidence, epidemiology, and ecology of cancer in Westmorland County, England,[12] is of interest. This survey, made under the auspices of the Westmorland Field Commission for Cancer Research, was carried out under the direction of Dr. Louis Sambon, whose cancer investigations in the American tropics, in Iceland, Holland, and Italy, are well known. This report states:

"In the first place, it was decided to discountenance all theories concerning causation, and to ignore, for the time being, all views based solely on clinical or pathological data. It seemed better to approach the disease as if totally unknown and collect only sheer facts . . .

Our investigations indicate very forcibly that we are confronted with a systemic infection characterized by long latency and exhibiting varied local manifestations, determined in type and site by the most diverse physical, chemical, mechanical, or animate irritants. Insidious invasion, long quiescence, complexity of secondary factors, muteness of symptoms, and protean nature of manifestations are the peculiar features which have baffled inquiry so persistently.

Dr. Sambon believes that *cancer* is brought about by the activities of an exceedingly minute endocellular parasite strictly adapted to its specific cell host. He points to *Rickettsia*, the germ of typhus, which inhabits almost exclusively the endothelial cells of the capillaries, giving rise to marked defensive proliferation, the formation of intravascular overgrowths (Fraenkel's "nodules") and metastases. He looks upon the gross tumour—the actual *cancer*—as a late, often fatal, manifestation of the cancerous infection not unlike the gumma in tertiary syphilis. As in tuberculosis, so also in cancer, the specific germ appears to be universally prevalent and of wide zoological distribution.

Perhaps the most striking, the most interesting discovery our investigations have elicited is the great prevalence of all types of malignant and other neoplasms in animals. We have met with cancer in all kinds of wild and domestic animals, in pet animals, and in animals used as food. We might mention horse, cow, sheep, hog, cat, dog, hedgehog, rabbit, vole, rat, mouse, fowl, turkey, goose, owl, canary, and toad. Fowls of all varieties (Leghorns, Wyandottes, Plymouth Rocks, Bantams, Anconas, Rhode Island Reds in our series) are particularly liable, and we have noticed small outbreaks of the disease in certain poultry flocks similar to the cage epidemics of rats and mice mentioned by Borrel in France and by Gaylord in America. This unexpected, remarkable prevalence of the disease in all kinds of animals opens up a wide field of possibilities which we are already exploiting."

In his lectures at the London School of Hygiene and Tropical Medicine, and in his epidemiological papers, Dr. Sambon has repeatedly stressed the importance of a full knowledge of the zoological distribution of disease. Without a knowledge of the animal sources of infection it is not possible to understand, far less to control, outbreaks of disease in man. A memorable example is that of Russian artillery sent to destroy all the inhabitants of a plague-stricken border village in order to stay the epidemic. The unfortunate people were

sacrificed, but no one troubled about rats, and the disease soon spread like wildfire. Is it not known now that many cases of tuberculosis are not of human source, but, on examination, prove to be of either bovine or avian type?

When once the organism which proves to be the cause of human cancer is discovered we should at once make a careful study of our domestic animals, especially dogs, cats, fowl, pigeons, mice and rabbits and especially raw vegetables and sources of water supply. If the organism is found frequently in these animals, then the proper steps for lessening the chances of infection from such sources should be taken. It is quite possible that some of these animals might be carriers of the disease without themselves being affected by it. It is possible that the organism may be soil organism and enter the system through uncooked vegetables or raw fruit.

Finally it might be possible to produce a vaccine or serum that would have an inhibitory or curative action upon malignant tumors, or would be of great prophylactic value when used after operation or in connection with irradiation.

These are some of the numerous and attractive fields of investigation that would be immediately opened up once the germ has been discovered.

To those who are thoroughly convinced that cancer cannot be due to a microorganism, this may all seem scarcely more than a beautiful dream. To those who are still open-minded, I have tried to show that the microbic theory of cancer rests upon a solid foundation of clinical facts and is further supported by a large and steadily increasing amount of experimental and laboratory evidence especially in the field of filterable fowl tumors. It will require much further work and investigation before this theory can be definitely established; but when we consider that this is a matter of such vast moment to the million or more sufferers from cancer and to the still greater number who are certain to suffer from cancer in the years to come, we who are engaged in cancer research, both clinical and laboratory, should feel stimulated to do our utmost to solve the problem of the cause of cancer. If the cause proves to be a specific living agent, as I believe it to be, then for the first time we shall have some satisfactory foundation for our hope of gaining control of the disease.

After you have listened to the arguments for and against the parasitic nature of cancer, you may feel yourself in the position of Rabbi Ben Ezra of Browning's poem:

Now, who shall arbitrate?
Ten men love what I hate,
Shun what I follow, slight what I receive;
Ten, who in ears and eyes
Match me; we all surmise,
They this thing, and I that: whom shall my soul believe?

Unlike Rabbi Ben Ezra, I freely admit that the question of the etiology of cancer is not so nicely balanced, and that I belong to a small minority. Still one may remember that in medical science as well as in politics, minorities have a surprising way of becoming majorities in a very short time. What we need most is more facts rather than opinions.

CONCLUSIONS

1. Every means, including those already adopted by the American Society for the Control of Cancer, should be employed to encourage early recognition of malignant tumors as well as prompt and proper treatment of them. This entails not only continuous propaganda to persuade the patient to consult a physician as soon as a tumor is discovered, but, and what is more important, entails greater effort in educating physicians in the difficult field of the diagnosis of malignant tumors.

2. A considerable portion of the funds raised for cancer control should be expended in the field of collecting more facts, especially more accurate firsthand information as to the geographical distribution of cancer, and especially, data that may account for the wide variation in

incidence; in other words, surveys should be made similar to the one of the Westmorland County, England, conducted by Dr. Sambon in 1926–1928.

3. Little advance may be expected in the surgical treatment of cancer, but in the field of roentgen-ray and radium, I believe, that, with increasing knowledge of the action of these agents, and with improved technique, considerable advance may be anticipated. Radiation has already displaced surgery in certain fields, i.e., cancer of the skin, of the cervix, and oral cancer. It is rapidly becoming recognized that most cases of cancer can best be treated by a combination of surgery and radiation, rather than by the use of either alone.

4. A further study of the treatment of inoperable tumors, especially sarcoma, by the toxins of erysipelas and bacillus prodigiosus, should be carried out. It is found that a considerable number of these cases beyond hope from any other method of treatment can be cured by toxins, and that in many operable cases in which the toxins have been used as a prophylactic after operation a larger percentage of patients have remained well for five years; therefore this method should receive more general adoption.

5. A systematic attempt should be made to give the undergraduate medical student much better clinical instruction in the early diagnosis of cancer than he has had in the past; and even more important is it to provide numerous centers where intensive post-graduate instruction may be obtained. Cancer departments should be organized in the larger general hospitals, to be in charge of men who have had wide experience in the diagnosis and treatment of cancer.

6. A number of so-called Cancer Institutes should be established in a few of the principal cities of the country, as recommended by Ewing at the meeting of the American College of Surgeons in October, 1929. Such institutes should have a large endowment to make them educational centers; they should be equipped to give the most approved methods of treatment; and they should contain research laboratories in which the whole field of cancer is studied.

7. The results of study by the various organizations and individuals working on the problem of cancer, including both the clinical and laboratory aspects, should be given wide publicity. At least every three to five years there should be an international cancer congress, bringing together the leading workers in cancer, and furnishing an opportunity for an interchange of ideas.

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HEMORRHOIDS—MEDICAL ASPECT*

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NEW YORK

THE object of this paper is to present the subject of hemorrhoids as observed in the practice of a gastroenterologist. The material utilized for this study differs from that of the rectal surgeon in that very few patients complain of hemorrhoids as their only, or even chief, trouble. As a matter of fact, the piles are for the most part either incidentally mentioned in the history, or incidentally discovered in the course of the routine physical examination. Although extreme examples of the disease are not encountered in this type of practice, it is nevertheless possible that a study of this character can give a fair notion of the incidence, etiology and management of hemorrhoids as they occur in general practice, or in the population at large.

PATHOLOGY

A few well-known facts may be recapitulated. Piles are localized varices of the hemorrhoid plexus (i.e., they are a form of varicose veins). Internal hemorrhoids arise from the submucous plexuses above the anal sphincter; the external variety arise from the subcutaneous plexuses outside the sphincter. Bleeding is more common from internal than from external hemorrhoids, and ordinarily follows the passage of hard feces. An "attack of piles" is usually associated with thrombosis. The latter condition is essentially an acute thrombophlebitis which results from trauma plus infection from within or without the rectum. Another common complication is prolapse of simple or inflamed piles into or through the anal canal. This may result in possible strangulation or gangrene. As attacks of phlebitis recur, the vein wall thickens so that ultimately spontaneous obliteration of the pile may take place with the formation of a tab of cutaneous tissue.

INCIDENCE

General: The incidence of piles in our material was carefully investigated. A review of 1892 records showed that piles were present on physical examination in 499 cases, an incidence of 26.3 per cent. Of these 499 cases, 170 showed active hemorrhoids; the remaining 328 revealed the presence of tabs. In other words, we may assume from *physical examination* that whereas only 9 per cent of all cases show active hemorrhoids at any one time, an additional 17 per cent of patients present tabs as presumptive evidence of nature's cure of this condition.

On the other hand the incidence of hemorrhoids according to the patients' *history* seems considerably greater. Of 786 individuals who were interrogated on this point, 382, or over 48 per cent, admitted that they were suffering, or had at some time suffered from this condition.

Sex: A group of 656 cases which showed hemorrhoids either by history or physical examination, or by both methods, was analyzed as to sex incidence. Of these, 392, or 60 per cent, were females, and only 264, or 40 per cent, were males. As these figures reverse the usual sex incidence as handed down in the literature, a further analysis was made of 329 cases in which neither the history nor the physical examination revealed the presence of piles. In this group, 57 per cent were males and 43 per cent were females. Now, since our general material has been carefully checked as to sex incidence and since the variation between males and females does

* Read before the Section of Surgery, New York Academy of Medicine, February 4, 1931.

not exceed 2½ per cent in groups of successive thousands, we feel safe in concluding that women suffer from piles more often than men in the proportion of 3 to 2.

Habitus: Of the 656 hemorrhoid-positive cases, 39 per cent were of the sthenic or stocky habitus and less than half as many (18 per cent) of the slim or asthenic build. This is to be contrasted with the general incidence of 27 per cent sthenics and 21 per cent asthenics in an unselected series of 3000 consecutive cases. Thus it appears that piles are associated with a stocky build almost twice as often as with the slim body habitus.

ETIOLOGY

Both predisposing and exciting factors operate in the production of piles. In regard to the former it has rightly been pointed out that the rectal veins possess no valves and that a considerable column of blood is supported by this vascular apparatus in the erect posture. A more important factor in this connection is the possibility that the venous plexuses in certain individuals are so rich and at the same time so weak that they congenitally predispose their owners to pile formation. This theory we believe, is somewhat supported by the greater incidence of hemorrhoids in sthenics mentioned previously, inasmuch as this build is known to be predisposed to certain other forms of herniation involving the digestive tract.

Among the exciting causes of piles, child-bearing and constipation are usually mentioned. We are inclined to agree as to the rôle played by the former factor, since many of the women questioned attributed their hemorrhoids to the birth of their children. As regards the influence of constipation, a special study was made because of the importance of this feature. To begin with, it is easy to be misled in this connection because constipation is a very common complaint. As a matter of fact, constipation was encountered in 54 per cent of our 656 hemorrhoid-positive cases. However, further investigation revealed the fact that almost the same proportion, viz., 52 per cent, of our 329 hemorrhoid-negative cases were constipated. On the other hand, although 28 per cent of 500 constipated patients showed hemorrhoids on physical examination, it must be recalled that 26 per cent of our 1892 unselected cases showed hemorrhoids under the same conditions. To make these facts more impressive, an analysis of 500 patients suffering from colitis, a condition associated with diarrhea rather than with constipation, showed an incidence of hemorrhoids of 29 per cent, i.e., slightly greater, if anything, than the constipated group. In other words, it seems clear that constipation as such has little if any bearing on the production of hemorrhoids. This is in accordance with the significant statement by many patients that their piles do not bother them so much when their bowels are "tied up."

In sharp contrast with this are the facts concerning the cathartic habit. A review of a series of 405 cases of cathartic habit showed that hemorrhoids were present on examination in 144 or 38 per cent, an incidence higher than in any other group studied and almost half again as great as the general incidence of 26 per cent. We are therefore in absolute accord with the opinion that it is the abuse of cathartics, and for that matter, of enemas and irrigations as well, that constitutes the most important cause of hemorrhoids.

The mechanism involved appears to be the following: The rectal and anal mucosa is accustomed to the presence of formed feces, even when these are hard and expulsion difficult. A fissure at most results from the added effort. When however, undigested food products or fluid feces are rushed down, often containing an irritating cathartic as well, the mucosa becomes inflamed, congested and edematous, and protrudes more and more into or through the anal canal with each succeeding violent or explosive effort at defecation. Instead of the anal canal hold-

ing firm and moulding the fecal column as in the orderly process of normal defecation, the entire engorged mucosal ring at or above the internal sphincter assumes the rôle of a foreign body, undergoes partial extrusion, and thus increases venous stasis and promotes varicose formation. In short, it is the abnormal and frequent defecation resulting from artificial purgation that "brings down the piles," to adopt a common form of expression.

SYMPTOMS

It is unnecessary to elaborate on the clinical picture of hemorrhoids, as it is well known to everyone. Thrombosis is always characterized by pain, and prolapse by pain, tenesmus, itching and discharge. Bleeding is usually slight but in some cases long continued small blood losses may lead to a severe anemia of the aplastic type with signs of bone marrow exhaustion (poikylocytosis, absence of erythroblasts, diminution in neutrophile and eosinophile leucocytes). Two such instances were observed in our series.

In 1 or 2 cases seen in consultation for suspected intestinal obstruction, the condition seemed to be aggravated by the presence of large prolapsed hemorrhoids, possibly induced by violent attempts at restoring bowel function. As soon as the ectopic varices were replaced the passage of gas quickly relieved the distention.

DIAGNOSIS

For the diagnosis of hemorrhoids, the left lateroprone position is useful. External hemorrhoids and tabs are readily seen on separating the buttocks. The recognition of internal hemorrhoids, however, is more difficult, and usually requires more special procedures. The finger introduced into the rectum may feel the soft irregularities or pouches just at or above the internal sphincter, but these may be empty of blood and not apparent except to the experienced palpator. A good technique for the demonstration of internal piles is to ask the patient to strain down as the examining finger is withdrawn. This extrudes the piles into, or even out of, the anal canal. A similar demonstration can of course be made on the withdrawal of any of the endoscopic instruments.

The chief differential diagnosis of hemorrhoids is from fissure. This should offer no difficulty if the buttocks are well separated and the anus carefully inspected while the patient is straining.

There should be no question of differential diagnosis between cancer of the rectum and hemorrhoids. Mistakes are made only because the finger is not introduced into the rectum.

TREATMENT

The treatment of hemorrhoids involves two aspects, management of the underlying condition and care of the acute manifestation. As regards the former, the first and most important task is the restoration of normal colon function. This is equally essential whether the patient suffers from constipation or from diarrhea. Restoration of normal colon function implies the spontaneous production of one formed stool approximately once a day. The technique of conservative bowel management has been described in detail elsewhere, but it may be well to repeat here that the first step is the absolute withdrawal of irritating cathartics and of the usual forms of local treatment with enemas and irrigations. At the most, a small injection of oil may be used nightly for a time in the treatment of rectal constipation. For this purpose a hand syringe with a hard rubber tip is employed, never a soft rectal tube, as this causes much more trauma.

In general the patients do best on a bland diet with or without the addition of some substance such as agar or psyllium seeds, or barium sulphate to furnish bulk without undue irritation. Butter, cream and olive oil are particularly useful in thin people, whereas mineral oil or oil agar mixture is better for the obese.

The treatment of the local condition

may be described as conservative or radical. The majority of cases do well on the former. Perhaps the first requirement is the proper toilet of the anus. Cleanliness and gentleness are essential. To this end, the patient should be advised to move the bowels at home whenever possible so as to insure adequate facilities for proper care. After defecation the anal region should be washed with non-irritating (castile or ivory) soap and warm water, using absorbent cotton for the cleansing. The parts should be wiped gently or patted dry with cotton, not with paper, and finally powdered with talcum.

If hemorrhoids tend to prolapse, it is imperative to replace them *immediately* after each extrusion. It is remarkable how many people go about with prolapsed hemorrhoids when all that is needed is the proper advice and a finger cot and some lubricating jelly. By taking this simple precaution early, much trouble can be avoided. Should this prove impractical with the patient up and about, he should be put to bed. At bed time a tannic acid suppository should be inserted or a salve containing this drug applied to the anal region.

If there is much pain and edema, bed rest in the latero-prone position is indicated. The application to the anus of cold compresses soaked in witch hazel often suffices for relief. In more severe cases, a suppository containing anesthesin, 5 grains should be inserted before defecation and one of bismuth with or without adrenalin after each bowel movement and at bed time.

The results of this method of management, which I believe is that usually practiced by internists, are on the whole satisfactory. Relatively few of our cases have required surgical therapy.

On the other hand we believe surgery is clearly indicated in the following circumstances:

1. Hemorrhoids that are responsible for severe anemia. Here the operation should be preceded by blood transfusion.

2. Large hemorrhoids that are irreducible, or those that prolapse despite repeated reduction.

3. Ulcerating, gangrenous, or strangulated hemorrhoids.

4. Thrombosed hemorrhoids, although in patients unwilling to be operated on, and satisfied to bear the pain for two or three days longer, satisfactory results can be achieved by expectant treatment. In such cases, there is spontaneous absorption or expulsion of the blood clot.

The results of past surgical procedures as observed in our cases were investigated and may be summarized as follows:

Some form of radical operation was performed ninety times on 80 patients so that 12 per cent of our series of 656 hemorrhoid-positive patients were operated on. In addition, 4 patients were treated by injections and 2 by electrolysis. The injection cases did well, but one case treated by electrolysis resulted in failure.

The 90 surgical operations were followed by return of hemorrhoids in 22; by stricture in 8, by bad scar causing pain, spasm or bleeding in 3; by fistula in 1; and by paralysis of the sphincter in 1, a total of 35 cases or 40 per cent of questionable results in this series. In our opinion, however, the 22 cases showing return of hemorrhoids should not necessarily be charged up to poor surgery, since if our hypothesis that hemorrhoids are due to improper bowel hygiene is correct, there is no reason to expect permanent immunity from hemorrhoids if the original perpetuating factor remains active following the operation. After all, a surgeon operates for a lesion that exists, not for a lesion that may develop. Indeed, a radical excision of the entire ring of hemorrhoidal plexuses would result in a mutilation far worse than the original disorder. Accordingly, subtracting the 22 return cases, we have a net residue of 13 cases, or 14 per cent unqualified surgical failures.

It is hardly necessary to add that surgery in itself is not a complete treatment for piles, any more let us say, than it is a complete treatment for gastric ulcer. The original predisposition and the possibility of continued functional mismanagement still remain to invite the continued supervision of the experienced internist.

[For Summary see Author's Reprints.]

SURGICAL TREATMENT OF HEMORRHOIDS*

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SURGICAL treatment of hemorrhoids is not new. This operation has been routine for hundreds of years, but advances have been made in technique and in improved surgical principles. If we have not invented a new operation, we have at least been so fortunate as to learn what to avoid.

For some unknown reason a rough job seems to have had the right of way, whereas only the gentlest handling in this sensitive area should be allowed. Some of the archaic operative methods in vogue when I first became interested in rectal work still survive. I admit I was myself responsible for a form of torture by the invention of a plug, sufficiently large to make anyone's postoperative convalescence a hell on earth. With a powerful divulsion of the sphincter before operation, and the use of this plug afterwards, the operation was one never to be forgotten by the patient. They say the thumbscrew and the rack were playful in comparison. I have done all I could to counteract the misery I set forth, but it still seems to have a fascination for some operators, as news of it drifts back to me occasionally. I wish now to go on record that I condemn the plug I invented.

The operation as practiced today, under local anesthesia, is simple, effective and practically painless, and the patient is not detained in the hospital for more than four or five days.

I cannot too strongly condemn the old practice of divulsing the sphincter. It is crude; often followed by incontinence and invariably causes severe pain, hemorrhage into the muscles, and often a certain amount of dysfunction. If the internal and external sphincters are over-developed or spastic, it is safer to cut them posteriorly. If incontinence should result a plastic operation will remedy this misfortune, but there is no remedy if incontinence follows divulsion.

Hemorrhoids are so often associated with other pathology that they should only be treated after a thorough proctoscopic examination satisfies the surgeon that there is no complication. About one-third of patients suffering from cancer have had a hemorrhoidal operation without this more serious condition being noted. This lack of discernment also applies to infection of the colon, diverticulitis, polyposis, and internal fistula, which Dr. Kantor has mentioned. Probably the infection was present before the operation and was not discovered. I can readily see the great importance of a thorough examination.

The frequency of hemorrhoids is such that the layman imagines all rectal troubles are hemorrhoids, and often the explanation given by the patient is accepted without trouble by the physician. Bush, in 1836, emphasized the fact that hemorrhoids were only too often associated with more serious ills. Ninety years later we give voice to the same warning.

Uncomplicated hemorrhoids give no symptoms except a vague feeling of fullness, and indefinite discomfort with some loss of blood. Severe bleeding occasionally occurs into the bowel but it is rare. On the other hand, repeated small hemorrhages result in a secondary anemia which is brought to the patient's attention by shortness of breath and general weakness. Of importance are the secondary and deferred symptoms, such as digestive disturbances, loss of appetite and constipation. Mental depression is almost always associated with pathology in the anal region, particularly in hemorrhoids. If

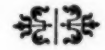
* Read before the Section of Surgery, New York Academy of Medicine, February 6, 1931.

these have existed for years with marked prolapse, loss of muscular tone is found, but one should be on guard not to confound this with a similar condition found in tabetics.

Today's operations are done nearly entirely under local anesthesia. Some men prefer spinal or general, but I like the local anesthetic better. Forty-eight hours before, a cathartic is given. In the evening an enema is given. In the early days I preferred the clamp and cautery to all other methods, but now I prefer the ligature. Perhaps for the man who operates infrequently, the clamp and cautery is safer. Surgeons have personal preferences, however, and by confining themselves to one method develop better technique. I achieve good results with ligature. All bleeding points, either in the hemorrhoidectomy or any other operation, should be clamped and tied and there is no fear of after-hemorrhages. The sphincter is cut posteriorly, and the wound packed with a little vaseline gauze. This remains in until after the bowels have moved which is generally forty-eight hours. My own practice is to give an enema of magnesium sulphate, 1 oz. to a pint of water at a temperature of 112°F., the next day followed by a mild cathartic. The patient is then given an enema and the gauze removed. This should be done very gently. The wound must be irrigated after each movement.

The patient is kept under observation without any additional physical interference until the wound is healed. The operation is practically devoid of pain. Even with the first movement, which is usually dreaded by the patient, he is agreeably disappointed.

To summarize: in the past, hemorrhoidectomy was not alone attended by pain and discomfort, but often by incontinence. Bad news travels fast and leaves a lasting impression. The dread of this operation which is still inherent in the minds of the general public may be founded upon the results which attended the procedure as formerly performed. I am frequently asked: "Is there any danger of losing control if this operation is performed?" In the average surgeon's practice there is at present very little danger of this occurrence.



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* Continued from p. 604.

HEMORRHOIDECTOMY BY MEANS OF THE HIGH FREQUENCY CURRENT*

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A METHOD for the removal of hemorrhoids which is by no means ideal but is one that has given good results over a period of several years and in the hands of a large number of men, is the application of the high frequency current. The probability is that, with a growing acceptance of the value of these currents when employed surgically in other portions of the body and with a better understanding of the application of these currents, the method will come into more general use.

In the effort to learn of the experiences of men who have been using the high frequency current for the destruction ot hemorrhoids, a questionnaire was sent out to which twenty-five relatively complete replies were received. This questionnaire with the summary of replies received, is as follows:

QUESTIONNAIRE ON THE USE OF THE HIGH FREQUENCY CURRENT (SURGICAL DIATHERMY) FOR THE DESTRUCTION OF HEMORRHIODS

1. For how long a period of time have you been using this method? Varied from six months to ten years.
2. How many cases have you operated on in in this manner? Totalled 3284.
3. Do you perform this operation in your office? 16 physicians.
   hospital? 2 physicians.
   both places? 7 physicians.
4. After the operation how long do you keep the patient in
   (*a*) the office? Not at all, up to two hours.
   (*b*) the hospital? Up to one week.
5. What instructions are given to the patient with reference to

	before operation	*after operation*
(*a*) food?	light diet	light diet
(*b*) enema?	usually	
(*c*) cathartic?	not regularly	mineral oil

6. What type of anesthesia do you use?
   (*a*) in office? Local, novocaine, butyn, procaine, quinine urea hydrochloride.
   (*b*) in hospital? Gas oxygen and ether.
7. What, if any, measures do you use in conjunction with the employment of this technique? None specially.
8. In what types of hemorrhoids do you use this method? Practically all types.
9. Results:
   (*a*) End-results? Good in every instance except one in which there is a report of stenosis in operation of first 5 cases.
   (*b*) Immediate results
      1. pain after defecation? Usually little, occasionally moderate, lasts three to ten days, usually about four to seven days.
      2. pain other times? Rarely.
      3. bleeding, at time of operation? Very slight or none.
      4. bleeding after operation? Occasionally, slight.
      5. for how long a period of time is the patient usually incapacitated? From no time at all to one week, very rarely longer.
10. Is your diathermy machine one of low voltage (up to 3500) or one of high voltage (beyond 3500)? Used about in equal proportion.
11. What methods of applications of the high frequency currents for destruction of hemorrhoids do you employ?

(*a*) Electrocoagulation (bi-terminal, d'Arsonval type of current).

   1. with one large dispersing and one active pointed electrode?
   2. Bierman clamp?
   3. other modifications?

* Read before the Section of Surgery, New York Academy of Medicine, December 17, 1930.

In every instance one or both of these methods.

(*b*) Electrodesiccation (monoterminal, Oudin type of current)? Occasionally.

(*c*) Fulguration (sparks? Not at all.

(*d*) Electric cutting current? Occasionally in conjunction with electrocoagulation.

(*e*) What is your opinion as to the relative place and merits of these high frequency methods as compared to each other? Coagulation preferred, particularly when applied with the Bierman clamp.

12. How does the use of the high frequency current methods compare with:
(*a*) the clamp and cautery operation?
(*b*) the ligation and excision operation?
In all but 1 instance, where the physician claims stenosis resulted from the use of coagulation, adding that this was due to faulty technique, etc., the use of the high frequency currents is considered distinctly superior to the clamp and cautery and ligation and excision methods.

13. Have you noted the effect of the use of the high frequency current on other rectal conditions found present along with the hemorrhoids, e.g. polypi, fissures and prolapse? Yes, in 12 instances, applied to polypi and fissures.

The essential facts to be noted in the questionnaire are as follows: That the group of doctors answering the questionnaire numbered 25; that they have been using the method during an interval of time covering six months to ten years, and that they have operated on a total number of 3284 patients.

The operation is usually performed in the office (23 out of 25). From this we may definitely conclude that the procedure is essentially an office one. The patient goes out of the office directly after the operation or may remain for a couple of hours. Patients operated on in the hospital remain there not at all or may stay for a period of a week. Patients are ordinarily advised to partake of a light diet before the operation and are told to have the lower bowel clean. To insure this latter condition they are advised to take an enema. Occasionally they are asked to take a cathartic. After the operation the usual advice appears to be that they continue the light diet and that they take some mineral oil.

The operation is in nearly every instance performed under a local anesthesia. The anesthetic is injected into the base of the hemorrhoids, perianally and through the sacral route. The local anesthetics used are novocaine, procaine, butyn and quinine urea hydrochloride. In hospital cases use is made of gas oxygen and ether.

As to the types of hemorrhoids destroyed by this method, practically all types were mentioned, external, internal, complicated and uncomplicated. Some prefer to employ it in one type, others, in another type. Exception is taken to its use in very large gangrenous hemorrhoids. It is obvious that the technique may be modified depending upon the conditions found, e.g., in thrombosed hemorrhoids when it is possible to incise first, turn out the clot and then destroy the hemorrhoid bed. In small skin tabs preference may be given to the use of the dessicating current inasmuch as coagulation gives a greater amount of after pain when applied to the skin. The complete unanimity of opinion, that the end-results are good, is the interesting comment in the answers to this question. Exception is only made in the report of the first five cases performed by one man who felt that this difficulty was due to faulty technique. One other operator mentioned the possibility of stenosis. It is of course obvious that there is no automatic limit which can be placed upon the extent of the tissue which is destroyed. The safest of procedures can do damage when recklessly applied. The pertinent fact remains that stenosis does not usually occur because of the slight amount and the flexibility of the resulting scar tissue.

The consideration of the final end-result is the important one. It is the criterion by means of which the value of a procedure must be determined. As to immediate results, the important question of pain

after defecation elicited the answer that it was slight or moderate, as a rule. Occasionally there was none at all and have been very moderate. In our own experience we have noted occasional bleeding after defecation with only 1 case of

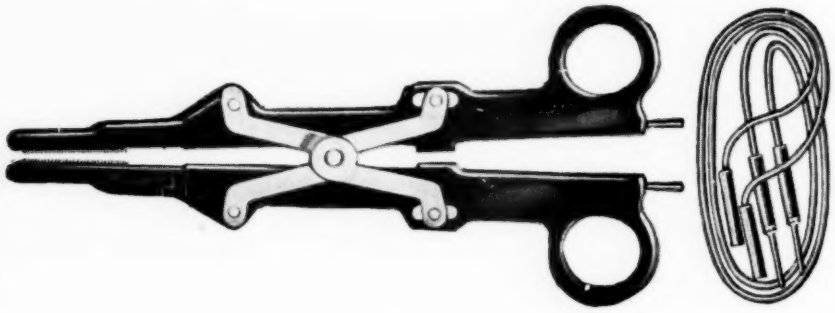

Fig. 1.

occasionally it was severe. When there is pain, it lasts from three to ten days, usually five. It is obvious that when tissue is removed or destroyed in this highly sensitive area, some pain should be expected. This can be greatly minimized at the time of operation if a few drops of 0.5 per cent quinine urea hydrochloride are injected into the tissues immediately adjacent to those destroyed. Radial incisions into the skin near the area of the destroyed hemorrhoid, made by means of a scalpel relieves the pressure of the subsequent edema, and thus prevents pain. The application of some ointment to the region helps to mechanically lubricate it; either simple vaseline or some preparation such as butesin picrate may be applied. To relieve the pain after defecation use may be made of a hot sitz bath or of hot, moist dressings kept warm by means of an electric heating pad pressed on top, the patient lying in bed, face down. Pain at times other than after defecation appears to be quite rare.

With reference to bleeding, the operative field is completely dry. Occasionally a few drops of blood are shed. The practically complete absence of bleeding appears quite dramatic to the surgeon, accustomed to seeing this as a bloody field. After the operation there is a serosanguineous discharge lasting several days. In the entire series, only 8 cases of secondary hemorrhage are reported and these appear to real secondary hemorrhage. This occurred at the crucial period (ten days after the operation) when the coagulated area was separating from the healthy tissue in a severe diabetic who had twelve violent bowel movements at the time. To my knowledge, this is the only case that required packing. The patient may be incapacitated for a period varying from one to seven days. Occasionally the period of incapacitation is shorter and it may be longer than a week. Naturally the length of time during which an individual will stay away from his work depends on several different factors, such as the extent of the hemorrhoidal areas destroyed, the psychologic reaction of the patient (with the extreme variations in pain-standing ability), character of the work performed, etc.

Judging from the answers to the questionnaire, high and low voltage machines appeared to be used in about equal proportion. There does not appear to be any great difference in the action of these two types of machines. In our own experience the electrosurgical hemorrhoidal clamp appears to work better with the low voltage machines.

Four distinct variations of the application of the high frequency current are available in the destruction of hemorrhoids. As far as our methods are concerned, we allow the character of the redundant

tissue to determine which one or combination of these methods is to be used. If there be but a small varicose area, the

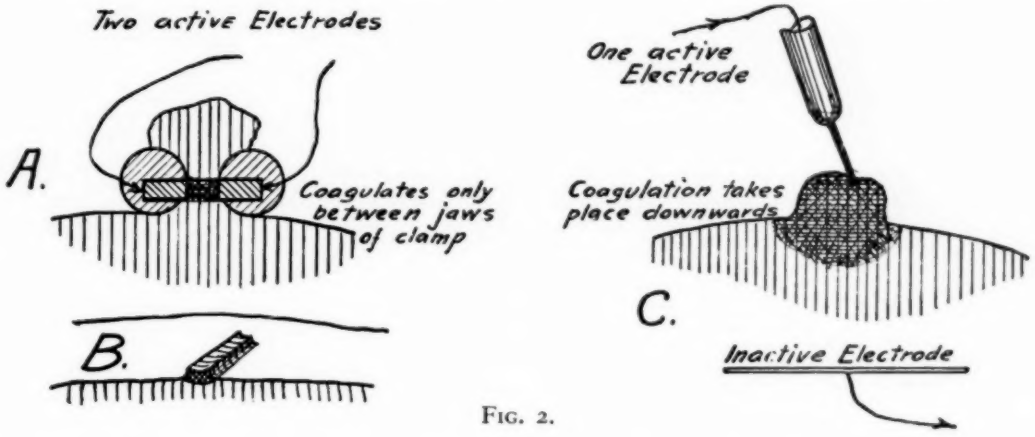

Fig. 2.

Oudin type of current is used, until a complete dehydration of the area occurs. We frequently intensify the action of this current by grounding. This is readily done by placing the patient upon a large metal electrode which is connected to a stem radiator or to a water pipe, etc. This method is called electrodesiccation. The destructive action of this current is not as intense as in electrocoagulation.

Fulguration consists in the showering of sparks from the tip of a pointed electrode held a short distance away from the tissue. The action of this procedure is a superficial carbonization. It is therefore but little employed.

The electric cutting current does not have sufficient coagulating power to seal the severed vein ends. It must therefore only be employed after the base of the hemorrhoid about to be removed, has been coagulated.

The procedure most generally employed is that of electrocoagulation. This current is a bi-terminal one with the same hook-up as that utilized in the usual medical diathermy, except that the current concentration is much greater so as to produce an actually destructive action. There are two methods of application: one is the use of a large dispersing electrode placed upon the skin in any part of the body, while the active pointed electrode rests on the area to be destroyed. The other technique is the use of a device consisting essentially of two active electrodes held in a clamp so as to permit of their synchronous application on either side of the hemorrhoidal tissue. Histologic examination of tissue

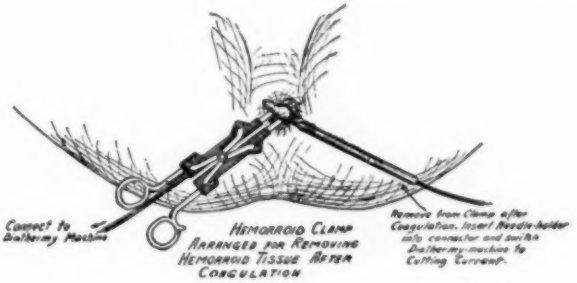
Connect to Diathermy Machine
Hemorroid Clamp Arranged for Removing Hemorroid Tissue After Coagulation
Remove from Clamp after Coagulation. Insert Needle-holder into connector and switch Diathermy-machine to Cutting Current.

Fig. 3.

destroyed by means of electrocoagulation shows hyalinization with complete loss of cellular outline.

We employ the pointed active electrode where the tissues cannot be grasped by means of the electrosurgical clamp. When using the pointed electrode a diagram of the electro-motive lines of force would represent a cone with its apex corresponding to the pointed electrode and its base to the dispersing metal plate. The depth to which the tissue is destroyed is dependent upon several variable factors which cannot be accurately gauged. These factors include the amount of current used, the length of time during which it is permitted to act and the electrical resistance of the tissues. The electrosurgical clamp is the preferable one in every instance

where it is possible to apply it because the destroyed area is more definitely delimited. The coagulation occurs only in the area lying between the jaws of the clamp. This obviates the danger of destroying too much or too little tissue. The uncertainty as to the depth of the destructive effect of the current is removed. The area is destroyed exactly as desired, permitting the immediate removal of the hemorrhoid.

The action of the electrosurgical hemorrhoid clamp is in no way comparable to that of the ordinary surgical clamp used in the clamp and cautery method. The ordinary surgical clamp compresses the tissue at the base of the hemorrhoid, insuring hemostasis when the tissue beyond it is removed. The electric clamp has only very slight compressing action, being so constructed that its jaws are separated where they are applied against the hemorrhoid. The very purpose of this separation is to prevent compression of the hemorrhoidal tissues, thus insuring a broad base which can be coagulated by the action of the electrical current. In the usual clamp and cautery operation, an artificial tuck of skin, of mucous membrane, or of both, is made and the tissue removed along a very thin linear area. This area is then seared over with actual cautery. What happens, therefore, is a superficial carbonization of tissues over a thin line. With this technique, the occurrence of secondary hemorrhage may be explained by the tendency of the adjacent remaining tissue, which has thus been artificially tucked, to pull apart, the gluing action of the thin and superficial carbonized area being insufficient to hold it together.

Using the electrosurgical hemorrhoid clamp, the destructive action actually occurs within the tissues embraced by the clamp. The resistance of the tissue to the passage of the electric current of high frequency, which is traversing it between the two active electrodes on either side, causes the development of heat sufficient to coagulate it. The destroyed area thus has sufficient width and depth to hold the healthy ends of tissue firmly together and to thoroughly cover the healthy tissue underneath.

After the base of the hemorrhoid has been coagulated, the area above it may be removed by means of scissors, scalpel, or the electric cutting current. This is better than permitting the tissue to remain in situ until it sloughs off ten days to two weeks afterwards, the dead mass acting like a foreign body, causing mechanical irritation and producing an offensive odor as it decays.

The action of the diathermy current differs markedly from that of the actual cautery in that the heat is produced within the tissues, due to the resistance which they offer to the passage of the electric current, while with the cautery, the heat is applied from an external source. In diathermy the electrodes do not become hot. With the cautery the applicator is red hot. This difference is a fundamental one, and accounts for the greater effectiveness of diathermy, and the particular value of its use in hemorrhoidectomy.

From these observations it is reasonable to conclude that the high frequency current is well adapted for the treatment of hemorrhoids.

A CASE OF
UTERINE FIBROID SEPARATED FROM ITS PEDICLE*

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FROM the small number of reported cases of uterine fibroids separated from their pedicles, this would seem to be a rare condition. I have been unable to find the report of a case found to have an acute separation.

Internal hemorrhage from pedunculated fibroids has been reported. Rokitansky[1] and Perrier[2] each reported a case in which there was a severe hemorrhage from a ruptured vein on the surface of a fibroid. Bruce Clarke[3] reported a similar case. Paul Zweifel[4] mentioned a case in which four days after a four months' miscarriage, a patient had a sudden and rather severe internal hemorrhage found to be due to a ruptured vein and artery on a fibroid. Lockyer[5] reported a case of hemorrhage from distended veins in torsion of the pedicle of a fibroid.

Cases have been reported in which there is complete separation of the fibroid from the uterus with the development of a new blood supply. Two such cases were mentioned by Lockyer,[6] and two by Cullen.[7] In these cases the new blood supply was from vessels in the omentum where it had become adherent to the tumors.

Wylie[8] reported a case of calcareous fibroma, detached from the uterus and rolled up in the omentum. It had no blood vessels entering it and the body tissues had treated it as a foreign body. A severe melancholia of previously undetermined origin was cured by the removal of this tumor.

A number of other cases of so-called "migrating fibroids" have been reported. They obtain their blood supply from the vessels of various abdominal structures and are entirely separate from the uterus. Examples of these are given in references twelve to eighteen.

Cullen[9] mentioned a case, reported to him by Dr. Bastenelli of Rome in a personal communication, in which the body of the uterus had spontaneously separated from the cervix. The woman had had several attacks of peritonitis. At operation the body of the uterus was found adherent to the anterior abdominal wall and entirely separated from the cervix. This had apparently been caused by a gradual torsion of the uterus by fibroids.

In all of these cases if the separation was acute it had occurred a considerable time before the operation. The more probable explanation is that the separation by torsion and development of the new blood supply was a very gradual process.

Cullen[10] wrote of a case of pedunculated fibroid which was shown to his students and examined by them immediately before operation. Upon opening the abdomen several hundred cubic centimeters of fresh blood were found in the pelvis and brisk bleeding was located as coming from a tear in the pedicle of the fibroid. He attributed this to the manipulations of the tumor during the examinations.

Maugeri[11] reported a case in which three very large fibroids were expelled from the uterus into the vagina, however without complete spontaneous separation from the uterus.

CASE REPORT

Mrs. M. K., a housewife, aged forty-two, entered Knickerbocker Hospital, Jan. 12, 1930, complaining of pain in the lower abdomen. While sitting in a theater the night before admission, she felt a sudden sharp pain in the lower abdomen. She then became quite faint. The pain continued after she reached home and had gone to bed. She was nauseated but

* From the Surgical Service of Knickerbocker Hospital, New York. Submitted for publication April 9, 1931.

did not vomit. By 5 A.M. the pain had become so severe that she called an ambulance.

Her entire past history was negative. Menstruation was always regular; the last period ten days before admission. She had two children, the youngest twelve years of age.

Physical examination was that of a well nourished and developed white woman about forty years of age who appeared acutely ill. The abdomen was symmetrical and slightly distended. There was no dullness in either flank. The entire abdomen was tender but this was most marked just above the pubis. Pelvic examination showed no discharge or bleeding. The body of the uterus could not be outlined. A large tender mass filled the pelvis. In the right fornix and posterior cul-de-sac was a distinct bulging which felt boggy. The bladder was distended but after catheterization the pelvic findings were unchanged. Also an area of dullness remained which extended about 3 in. above the symphysis pubis.

At the time of admission, patient's pulse was 80, temperature 97°F. and respirations 20. Blood count was red blood cells 4,000,000, hemoglobin 75, leucocytes 19,300, polymorphonuclear leucocytes 85 and lymphocytes 15. Urine was normal but for a trace of albumin.

Jan. 13, 1930. Pelvic findings were unchanged. Temperature ranged between 98 and 100°F. Pulse had increased to 112.

Jan. 15, 1930. The pain became much more severe, but her temperature remained between 99 and 100.°F. The pulse had increased, now ranging between 110 and 128. The tenderness and rigidity had also increased. The dullness above the pubis remained unchanged. The blood count was: Red blood cells 3,500,000, hemoglobin 67, white blood cells 24,200, polymorphonuclear leucocytes 92 and lymphocytes 8.

Because of the decrease in the red count and hemoglobin, the increase in the leucocyte count and pain, immediate operation was decided upon. A diagnosis of pelvic abscess or ruptured ectopic pregnancy was made.

Pathological Findings: An exploratory needle introduced into the bulging posterior cul-de-sac produced fresh blood. Upon opening the abdomen a large amount of bloody fluid was found. The pelvis contained fresh bright red blood and many clots. A large necrotic fibroid lay in the lower abdomen, resting on the brim of the pelvis. It measured 8 by 8 by 6 in. and was completely wrapped in omentum and loops of intestine excepting for a small area anteriorly, which was adherent to the anterior abdominal wall and bladder. The majority of the adhesions were fibrinous and separated readily. There were fibrous adhesions to a part of the omentum, one loop of ileum, and one small place on the sigmoid colon. These areas had the appearance of sarcomatous infiltration.

The tumor was found to be resting against, but not attached to its pedicle from which there was free bleeding. This pedicle was a flattened inverted cone measuring 2 in. across the raw surface. The apex was well down in the body of the uterus.

A hysterectomy was decided upon because of the possibility of malignancy.

Operative Procedure: With patient in lithotomy position the posterior cul-de-sac was explored with a needle. A lower midline incision was then made. This was later extended 1½ in. above and to the right of the umbilicus to permit delivery of the tumor. All of the adhesions to the tumor were freed and the resulting raw surfaces peritonized. A supravaginal hysterectomy was then done, the tubes and ovaries were left in. The raw surfaces in the pelvis were peritonized and the abdomen closed by layers without drainage.

PATHOLOGICAL REPORT

Macroscopic Examination: Specimen consists of a uterus without adnexa. It measures 7 by 4 by 4 cm. On section the uterine wall is found to be grayish in appearance, soft in consistency and irregular in thickness, varying from 2 to 4 cm. The uterus is ovoid in shape. On the fundus is an everted mass from the center of which protrudes a polyp 2 cm. in diameter. There is also a flat ovoid mass measuring 14 by 13 by 5.5 cm., soft in consistency, variegated in color, dark brown areas intermingling with grayish white areas. On section it presents some resistancy, hemorrhagic areas, and some that are yellow and brown.

Microscopic Examination: The uterus shows an increased number of muscle fibers and dilated blood vessels. The mass is a fibroid containing many necrotic and degenerating areas. The polyp is a polypoid adenoma of the uterus of endometrial origin.

Convalescence was uneventful. The wound healed by primary intention. The patient was discharged as cured on Feb. 3, 1930. The scar

was firm and pelvic examination negative. Examinations at intervals of three months since have shown her to be normal in all respects.

SUMMARY

A case of uterine fibroid is reported which at operation was found to be separated from its pedicle. This may or may not have been complete. It is readily possible that a remaining connection could have been separated while the fibrinous adhesions were being freed between the tumor and the surrounding structures.

The condition of the fibroid and the hemorrhage into the pelvis would indicate an acute severance of the pedicle of the tumor, at least so nearly complete that the blood supply was cut off. A peculiar condition was found in that the center of the pedicle was made up of a polypoid adenoma arising from the endometrium.

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HYSTEROSALPINGOGRAPHY

BY MEANS OF IODIZED RAPESEED OIL*

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ALTHOUGH the use of iodized rapeseed oil (campiodol) for roentgenographic exploration has been reported by Frazier[1] and by Glaser, Futch and Snure;[2] and emulsified campiodol has been used by Kutzman[3] as a pyelographic medium, no one as yet according to Witer, et al.,[4] has reported on the use of this oil in visualizing the uterine and tubal cavities.

FIG. 1A. Uterus and tubes injected with campiodol. Left tube is closed at cornual end. Right tube is filled to its distal end and is closed at this end, there being a mild degree of hydrosalpinx.

FIG. 1B. Film taken twenty-four hours after injection of uterus and tubes with campiodol. Note shadow of campiodol retained in hydrosalpinx. Same case as Fig. 1A.

Campiodol, as described by Frazier[1] has an iodine content of from 43 to 45 per cent, a specific gravity of 1.289 and relatively a low viscosity of 250 to 100°F. It deteriorates very slowly when exposed to light or heat. This iodized oil shows no toxic effects upon intravenous or subarachnoid injections. Frazier thinks that iodized rapeseed oil is as inert a material as can be obtained for this purpose and that it is non-irritating to the body tissues.

At the suggestion of Dr. Raiziss, who collaborated with Frazier and Glaser in the elaboration of campiodol,[5] I have used campiodol* in upwards of 30 hysterosalpingographies. I have made a special effort to observe these patients following the injection and I have found no ill effects whatsoever from its use. Moreover I find no disadvantages in the use of this oil and it has one strong advantage over other oils,[5,6] that I have used, namely, its low viscosity. Less pressure is needed for the injections, hence there is less time used per injection and less pain to the patient.

The shadow-casting property of campiodol, when injected into the uterus and tubes is highly satisfactory (Figs. 1 to 6).

TECHNIQUE OF INJECTION

Needless to say, in the injection of iodized oil into the uterus and tubes, one should exercise the principles of strict asepsis. The patient should receive an enema and empty the bladder prior to the

*This product consists of four parts campiodol and one part ethyl olive oil.

*Submitted for publication March 2, 1931.

injection, although failure to do this does not preclude good results. The patient is placed in the lithotomy position over a Bucky diaphragm with the film in place. A bivalve speculum is then inserted into the vagina and the cervix is exposed. The vagina and cervix are carefully wiped free of mucus, and the cervix and the external os are painted with tincture of iodine. The anterior lip of the cervix is then grasped with a volsellam and a sound is introduced into the uterus to determine the direction of the uterine cavity and its depth. (The larger the uterine cavity, the more oil is required.)

For injecting the oil, I use a modified Keyes-Ultzman urethral cannula with a rubber tip attached. The cannula has a soft pliable rubber acorn attached about 4 cm. from its end. When the cannula is inserted into the uterine cavity slight pressure on it causes the small end of the rubber acorn to fit snugly into the external os of the uterus and thereby stops leakage of the iodized oil from the uterus during the taking of the roentgenogram.

A syringe containing approximately 20 c.c. of oil is fitted into the cannula by means of a bayonet attachment. The cannula is then inserted into the uterine cavity in the direction made by the sound, so that the rubber tip actually plugs the external os and acts as a cork. If the oil should be too viscous, warming will make it flow more easily. Campiodol flows freely without being warmed. The oil is then injected slowly and carefully until approximately 3 c.c. have been injected. At this point the patient experiences slight uterine contractions and after a pause of a few seconds more oil is injected. The amount of oil injected is variable, depending, first, on the size of the uterine cavity, and second, on the amount of resistance felt. (A large or deep uterine cavity will hold more than 10

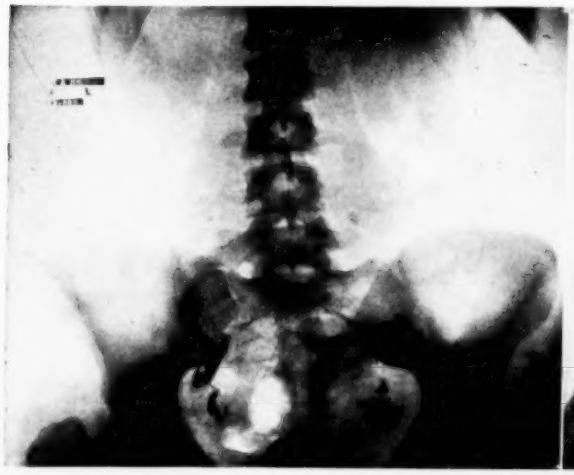

Fig. 2. Uterus and tubes injected with campiodol. There is marked spilling through distal end of each tube.

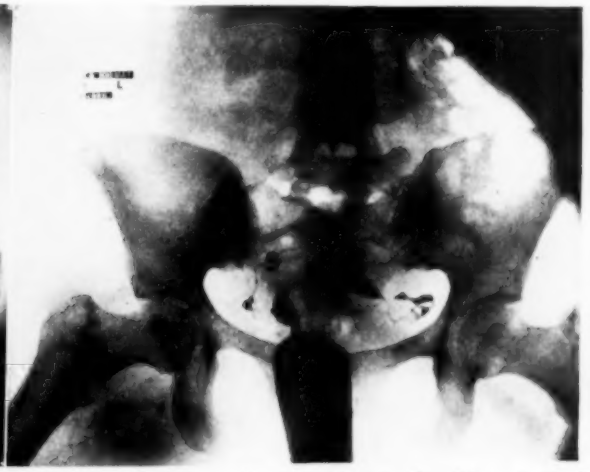

Fig. 3. Uterus and tubes injected with campiodol. Note elongation of uterus; stricture at internal os; marked deviation of fundus to one side; well-filled tubes with spilling at both distal ends.

Fig. 4. Uterus and tubes injected with campiodol. So-called sphincter of Kennedy is clearly visible at cornual end of tube.

c.c. of the oil and a small uterine cavity, with the cornual ends of the tubes obstructed, will offer marked resistance to the oil have been injected it is well to ask concerning the uterine cramps and to give assurance that they will soon pass off. In

FIG. 5. Bicornuate uterus injected with campiodol. This roentgenogram was taken through an enormous ovarian cyst.

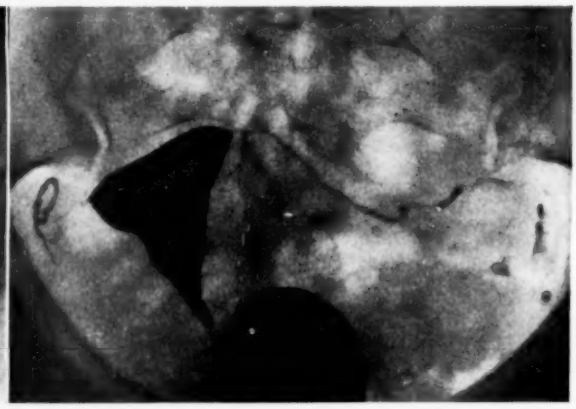

FIG. 6. Uterus and tubes injected with campiodol. Note stricture at internal os. Note conformation of tubes indicating former tubal infection.

over 5 c.c.) From 5 to 8 c.c. of the oil will fill the average uterus and tubes. When one estimates that the uterus and tubes are filled the injection is stopped, the cannula is held in position, the signal is given, and with the instruments in position, the flash is made.

There is no doubt that the use of the manometer is a refinement in the technique. Jarcho[7] has devised an instrument which is an excellent piece of apparatus and is extremely practical. I am absolutely convinced that a manometer of some type should be used to avoid the possibility of excessive pressure. The upper limit of pressure should be 200 mm. of mercury.

During preparation for the injection of the campiodol the patient is apt to become apprehensive. For this reason it is wise to explain in detail just what is to be done and in what manner the picture is to be taken. Warning should be given that slight uterine cramps will be felt and that any movement on her part after the procedure has started will necessitate an additional injection. Gentle admonition is wise before grasping the cervix with the tenaculum and again when the uterine sound is passed and the cannula is inserted. After 3 c.c. of my experience it has been only the neurotic patients who have suffered any inconvenience during or following the use of this diagnostic maneuver (in no patient have I been unable to complete the procedure). With the aid of a good light and accessible instruments the entire injection can be made, together with the taking of the skiagram, in less than two minutes from the introduction to the removal of the speculum. The patient is then turned on her side and a lateral view taken. This makes for additional precision. A part of my routine consists in the taking of twenty-four-hour plates to determine the possible presence of leakage into the peritoneal cavity where the tubes have been thought to be closed at the first examination. This procedure may be of further value in demonstrating the retention of the opaque substance in an hydrosalpinx which is permeable at the cornual end. I have operated on many of these patients following the injection of campiodol. I have found no evidence whatsoever of peritoneal irritation following the injections, and no signs that the campiodol was at all toxic to the peritoneum.

[For References see p. 642.]

STAPHYLOCOCCEMIA FOLLOWING CESARIAN SECTION: RECOVERY

REPORT OF A CASE WITH A RARE ANATOMICAL ANOMALY*

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THIS case is being reported on account of its many unusual features, the most outstanding of which are:

1. A rare congenital anatomical anomaly, necessitating a cesarian section.

2. An unusual type of infection entering the blood stream, with positive cultures recovered from the blood and other body fluids.

3. The extreme hyperpyrexia and complications.

4. The treatment and recovery.

Mrs. F. H., twenty-two years old, married six months, gravida zero, consulted me on November 28, 1927, on account of not having menstruated during that month. She gave her menstrual history as having commenced to menstruate at the age of sixteen, of the thirty-day type, and four to five days' duration. She was always regular, and the periods were never accompanied by premenstrual comenstrual, or postmenstrual pain. Her last menstrual period occurred on October 1, 1927. She was, therefore, four weeks past her expected period.

She had employed contraceptives for the first few months of her married life, but discontinued them two months prior to the consultation.

Examination: Weight, 131 lb.; height, 4 ft., 11½ in.; chest and abdomen, negative and urine, normal. The external pelvic examination revealed a normal bony contour, with normal pelvic measurements.

The vulva presented a normal appearance, although the introitus was unusually relaxed and large, for a nullipara.

Introduction of two fingers into the vagina revealed some unusual and interesting findings. The vagina was unusually short, shallow and wide, its length from vault to introitus being about 1½ in.

There was complete absence of a cervix; nor could the examining finger locate an opening or depression where, normally, a cervix or entrance into the uterus should be. The impression conveyed, was, that the finger was in a shallow, cup-shaped, smoothly-lined cavity.

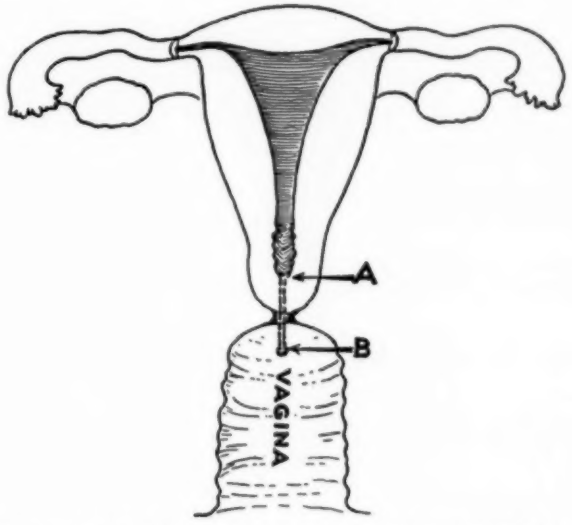

FIG. 1. Cervical anomaly as it appeared to examining finger. A. Lower uterine segment with very narrow canal into uterus. B. Pinpoint opening of canal into vaginal vault.

Bimanually, a uterus could be palpated, which was of normal size, easily movable, in good position, and apparently attached by its cervical portion to the vault of the vagina, intrapelvically. Adnexa could not be palpated. Rectal examination did not elicit any further information.

A speculum examination revealed the short, shallow cup-shaped vagina, with total absence of a cervix. A thorough search of every part of the vagina with a fine probe failed to reveal any opening into the uterus, or into any other adjacent structures. The patient returned two days later for further examination. The vagina was cleansed and dried, and under special illumination every part again examined with sound and probe. Again, no opening or depression could be located.

Owing to my skeptical attitude as to her menstrual cycle, the patient refused further

* Read before the Brooklyn Gynecological Society. Submitted for publication March 12, 1931.

examination. Exactly three months later she appeared again at the office, and to my surprise I found the fundus of the uterus one finger's

Hospital. Examination showed a head presenting, not engaged, dorsum to the right, fetal heart in the right lower quadrant, of good rate

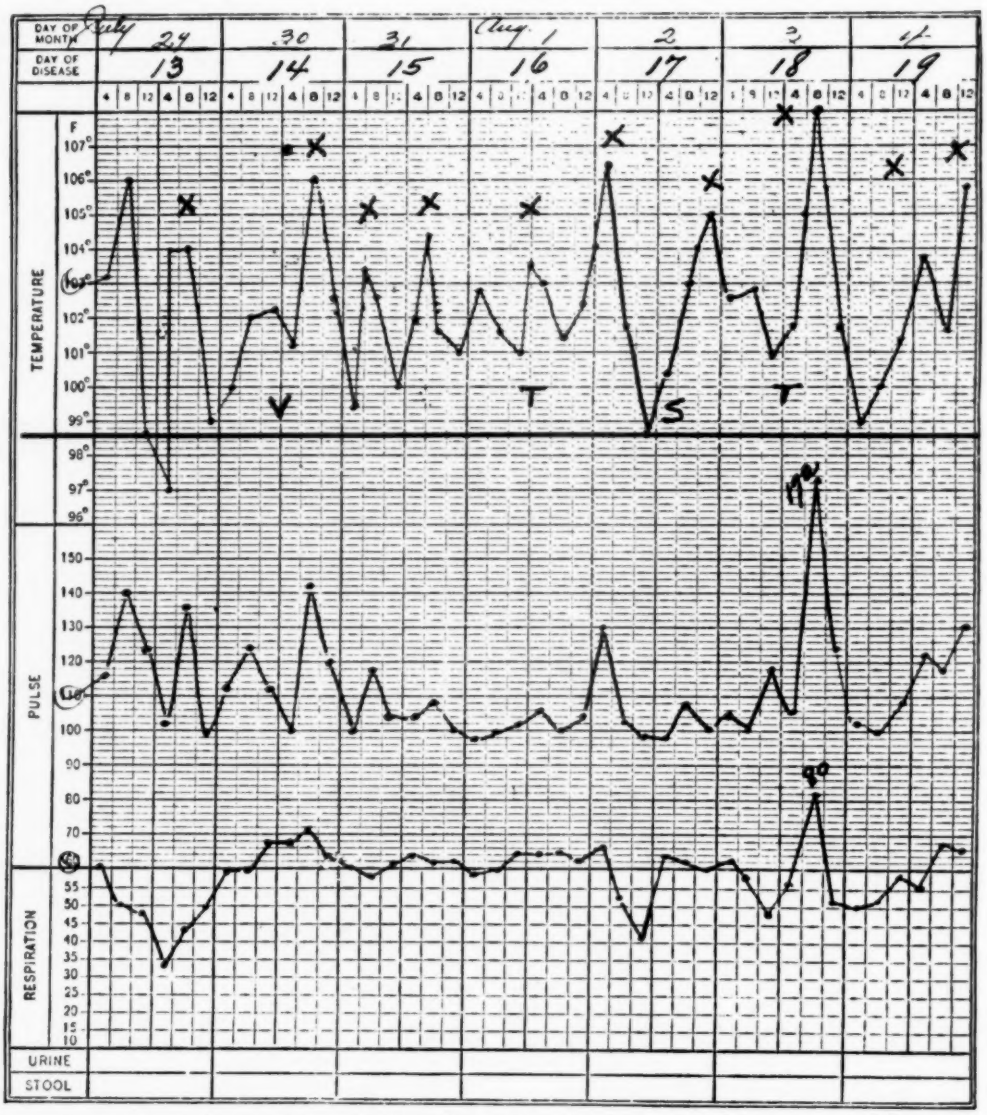

Fig. 2. Extreme excursions of temperature, high pulse and respirations, and frequency of chills. x. marks daily chills. v. Cultures taken at this time showed only 5 colonies after seventy-two hours. s. Cultures taken at this time were sterile after seventy-two hours. t. Transfusions.

breadth below the umbilicus. One month later, a fetal heart could be located.

Though I was now certain that the patient was pregnant, the puzzle was, nevertheless, not yet solved as to the opening into the uterus, as similar searches and probing were carried on at almost each visit to the office.

The patient's last menstruation was October 1, 1927; the estimated date of delivery was, therefore, July 8, 1928.

On July 15, at three P.M., the patient was admitted to the Brooklyn Hebrew Maternity

and quality. The patient complained of weak, irregular pains. Five hours later the pains were every five minutes and much stronger. Three hours later a vaginal examination was done. No opening of any kind could be located; but through the thickness of the vaginal vault a one-finger opening into the uterus could be made out through the intrapelvic portion of the cervix (if indeed, a cervix it was).

A cesarian section was immediately performed, and a living, normal male infant weighing 7 lb., 2 oz., was delivered.

The incision was made into the midline and lower half of the uterus, the incision encountering the placental site. Closure was effected with two layers of interrupted chromic catgut No. 2, and peritonealized with a continuous suture.

Examination of the uterus and appendages at the time revealed normal tubes and ovaries. It was not deemed advisable at the time to explore the pelvis for anatomical curiosities.

The patient was put to bed in apparently good condition, and a Harris drip instituted. Four hours later the pulse was 90, blood pressure 120/74 and respiration 40. The rapid respirations commenced at the outset and continued throughout the febrile course, ranging anywheres from 36 to 84 per minute.

The usual postoperative temperature set in, which gradually came down to normal by the fourth day. The lochia was sanguineous, rather scant, and rapidly changed to a grayish color by the fourth day, and became foul-smelling within another day. Concomitantly with this, the temperature commenced to rise again, reaching 103°F. on the fifth, and 105.2°F. on the sixth day. The respirations ranged between 40 to 50 per minute, and the patient became prostrated. Chilly sensations accompained the rise in temperature, but they were of brief

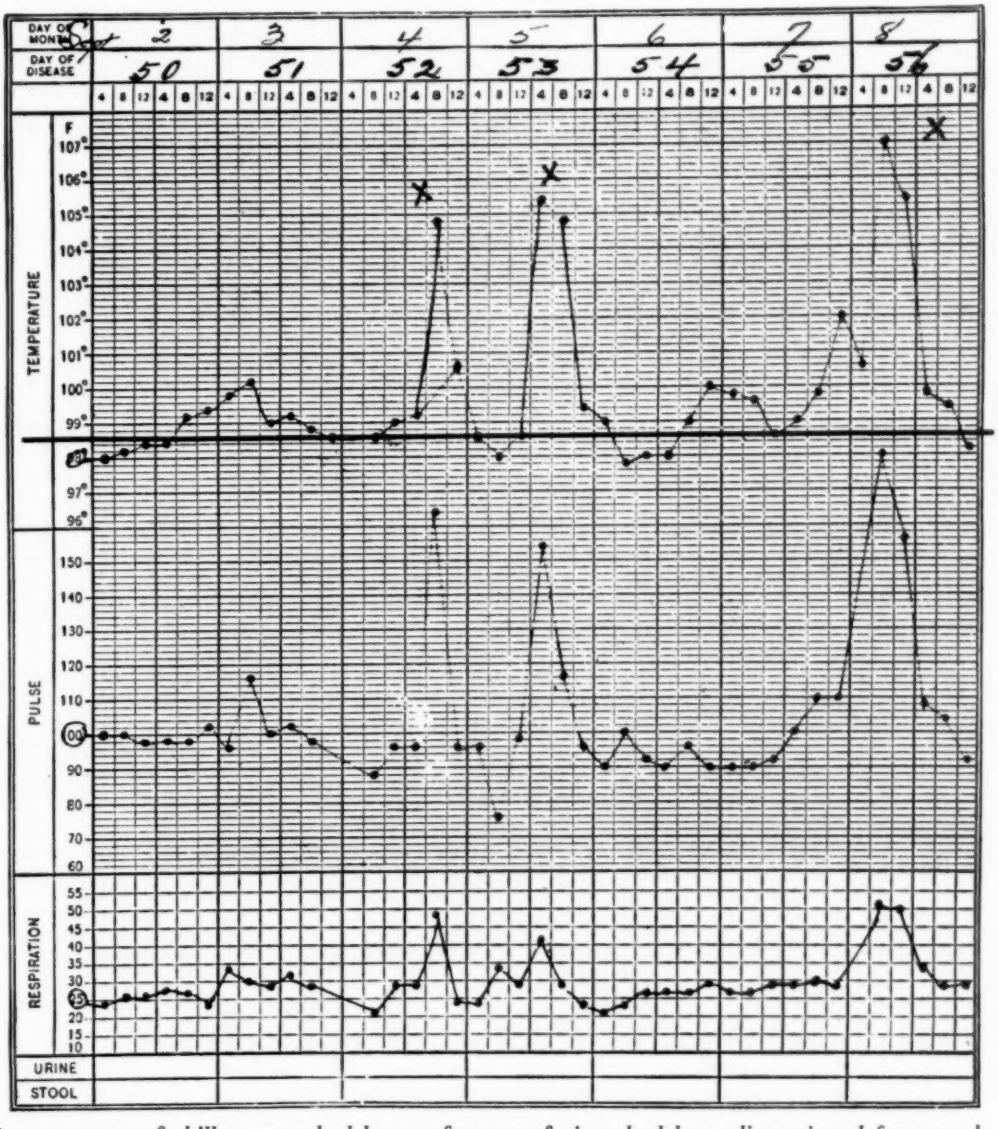

Fig. 3. Reappearance of chills, as marked by x, after transfusions had been discontinued for a week; also the rising again of the temperature, pulse and respirations.

duration. The lochia became definitely purulent and scant.

Examination of the patient at this time showed the abdomen to be moderately distended, and not tender to pressure. The operative wound was not indurated, and was healing by primary union. The urine was negative. A blood examination showed hemoglobin 40 per cent, red blood corpuscles 2,000,000; white blood corpuscles 18,000 with 90 per cent polymorphonuclears. She was of type 2 in the blood grouping. The blood was cultured.

There was no question by now that we were dealing with an intrauterine infection, due probably, to a lochiametra.

The patient was taken to the operating room and the vaginal vault again inspected. The vagina was cleansed and wiped dry. In a few minutes, a droplet of pus was seen to appear at the apex of the vault.

Into this, a sound was inserted, and then the point of an intrauterine dressing forceps. Considerable pain was caused by this maneuver; so a 1 per cent solution of novocaine was injected around this area, and the opening was enlarged sufficiently to permit a cigarette drain to be passed into the uterus. Within eight hours the uterus expelled the drain; so a perforated rubber catheter was inserted and sutured to the vaginal vault with silkworm sutures.

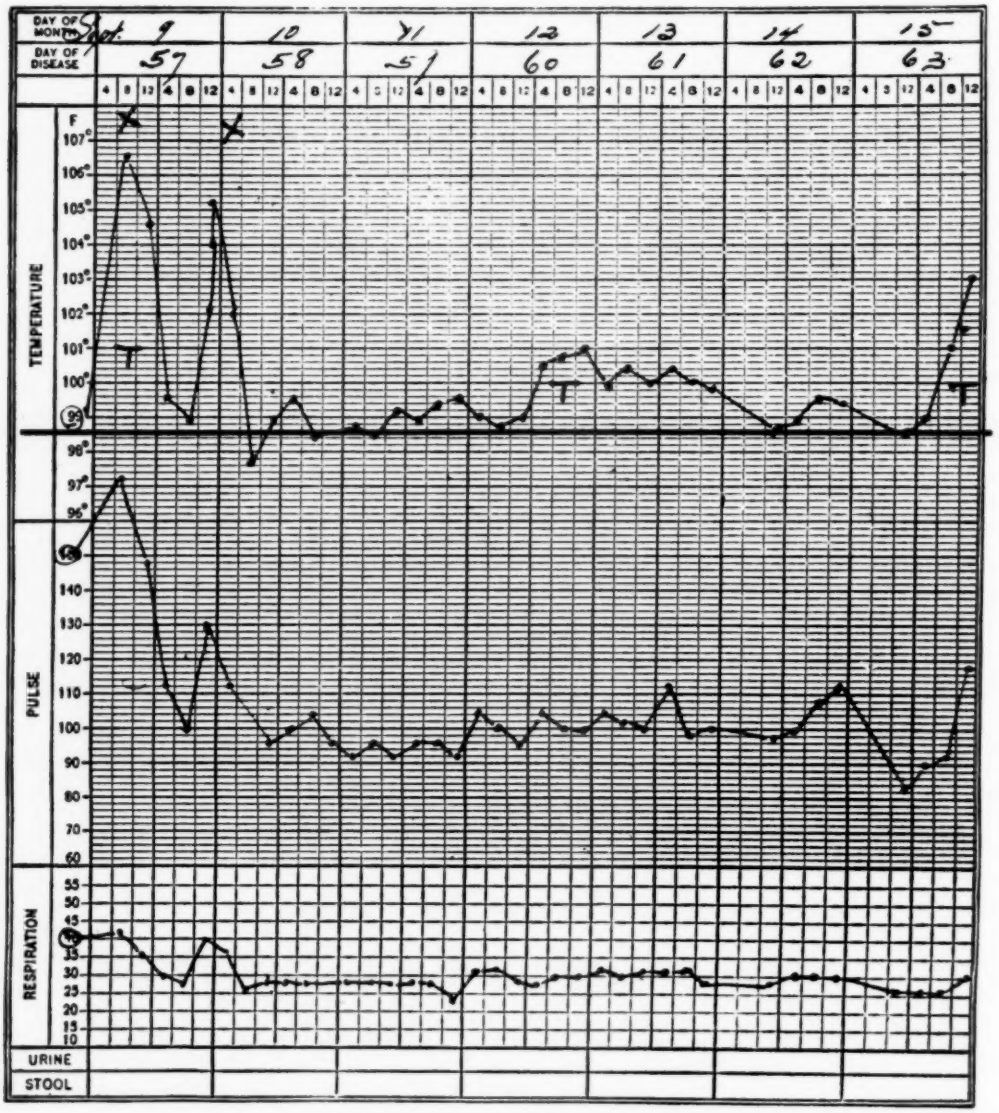

Fig. 4. Resumption of transfusions T, disappearance of chills, and falling of temperature, pulse and respirations. Chills never appeared again and patient recovered.

Pus drained through the tube very well; but within forty-eight hours the tube was expelled.

On the eighth day the patient suffered a severe chill lasting thirty-five minutes, the temperature rose to 106°F., and she appeared extremely toxic.

Dr. John O. Polak who examined the patient at this time, thought that she was suffering from a lochiametra with absorption, and the possibility of a pulmonary embolus that had occurred immediately after the operation.

Within thirty-six hours after the blood was cultured, the laboratory reported the presence of innumerable colonies of Staphlococcus aureus of the hemolytic type.

A transfusion of 150 c.c. of blood from a typed donor was given, with hopes of repeating it every other day. The patient's condition, however, grew worse the next day, and Dr. Polak was asked to see the patient again. An abstract of his opinion is as follows:

"Patient's circulation is failing, and her condition is desperate. Would *not* do any transfusions, but would inject intravenously, either rivanol or mercurochrome. Would also use the puerperal anti-streptococcus serum of Dr. Falls—Lilly."

This was done. The patient received 40 c.c. of the serum intramuscularly every day, for five days; and 2 intravenous injections of rivanol. The blood count remained the same; but a blood culture taken again on the thirteenth day postoperatively, after the patient had already received 4 doses of the puerperal serum, and 2 doses of rivanol, showed only 5 colonies of the Staphylococcus aureus after seventy-two hours.

Another culture taken four days later was sterile after seventy-two hours.

Despite this improvement in the blood stream infection, the chills continued unabated, the temperature ranged from 96 to 108°F., and the patient was in extremis almost daily.

The abdominal wound which had healed firmly by primary union, commenced to slough; marked edema of the vulva developed, as well as passive congestion with profuse râles, in the right lung. Within two more days this right side of the chest become flat to percussion, and breath sounds disappeared. A portable *x*-ray plate revealed the presence of a right-sided effusion probably encapsulated. Small areas of consolidation were also scattered in the left lung. Respirations were extremely embarrassed and ranged anywheres between 50 and 80 per minute.

A needle was inserted into the encapsulated area and 25 c.c. of turbid, yellowish fluid were aspirated. Three days later a similar amount was withdrawn. This fluid showed a pure culture of the Staphylococcus aureus.

The serum not being obtainable anymore,* we resorted to blood transfusions from donors that were immunized to the Stapylococcus aureus. The quantity ranged from 150 c.c. to 250 c.c., and was given every other day until the chills abated, then every third day. In all, the patient received one transfusion in the beginning, and then 12 transfusions from immunized donors, until all the chills disappeared, and until the temperature was below 102°F.

During this time the patient developed multiple abscesses over the abdomen, chest, thighs, etc., 14 in all. Two large Bartholin abscesses appeared at about the same time. All the abscesses were incised and drained. Cultures obtained from the abscesses contained the same strain of Staphylococcus aureus hemolyticus.

The lungs gradually cleared up, the abscesses healed, and the patient made a slow and tedious recovery. She was in the hospital three months.

Examination of the patient three months later showed the uterus to be of normal size, in second degree retroversion, and the adnexa negative.

The opening in the vault of the vagina was barely visible, and just admitted a uterine sound.

As to the Anomaly: A search of the current textbooks in obstetrics and gynecology, both in English and in German, fails to shed much light on this anomaly, except that it is mentioned as a rare malformation of the uterus.

Albutt, Playfair and Eden[1] refer to it as "uterus acolis," the total absence of the cervix, or "uterus parvicolis," a uterus with a rudimentary cervix. Penrose,[2] quoted

* This puerperal anti-streptococcic serum was prepared under the formula and direction of Dr. Falls of the University of Illinois. The serum was not on the market, but a limited supply was obtained by Dr. Polak who was kind enough to give us all he could spare.

by these authors, cites such a case. Bland[3] in the first volume of his "Gynecology" says: "sometimes the cervix is rudimentary or entirely absent." Schauta[4] says that the absence of the cervix is extremely rare; and Kermauner[5] in his exhaustive monograph on uterine malformations gives it but brief mention. Menge[6] simply mentions the possible absence of the cervix and places it in the proper order of the uterine malformations.

As to the Infection: It is common knowledge that staphlococcemia is not the usual type of infection following cesarian sections; yet such infections are by no means rare. This case, however, was particularly marked by its enormous severity, the extreme excursions of the temperature, the rapidity of the respirations, the frequency and durations of the chills, and the numerous complications.

As to the Treatment: There is no way of determining whether in this case it was the puerperal serum, the rivanol, or any other factor that caused the blood cultures to become sterile so early in the disease, though the chills still continued on; but I am confident that the blood transfusions of the immunized donors were a decided factor in the alteration of the blood stream infection, as shown by the disappearance, reappearance, and the final disappearance again, of the chills, when blood transfusions were again instituted.

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*Continued from p. 636.

FOCAL INFECTION*

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EVANSTON, ILL.

IN private practice prenatal care constitutes one of the best contributions to preventive medicine that the medical profession has developed. A great deal has been written lately about the need for teaching preventive medicine in our medical schools and various men have made the statement that even though our present-day physicians would attempt to practice preventive medicine privately, they would not have adequate training to make it a success. It is up to the coming generation, therefore, to develop and to establish preventive medicine in private work.

Obstetrics, not only in prenatal care but in all of its phases, is essentially preventive. It is not surprising, therefore, that we find preventive measures develop in obstetrics that are not seen in other branches of medicine. The average obstetric patient makes about twelve to fifteen visits to the doctor's office prior to delivery. During that time she is watched most carefully for the development of toxemia, anemia, obesity, miscarriage, thyroid toxemia, etc. At the same time she is given the benefit of various examinations. The care which has been developed so far, however, constitutes only a small part of what is possible to accomplish and among those things which we have somewhat neglected in the past, I feel sure that focal infection should be included. There have been no routine measures adopted by the average obstetrician to detect or clear up various foci of infection although a number of men have written very good papers stressing their importance.

Focal infection has been definitely proved to be an etiological factor in miscarriage, pyelitis, mastitis, phlebitis, anemia and toxemias of pregnancy.

Curtis,[1] in 1916, reported 2 cases where a mother's urine, placenta and fetal heart blood in still-birth yielded large numbers of streptococci which, when injected intravenously in pregnant guinea pigs produced abortion in every case and streptococci in pure culture were recovered from the uterus.

Nickel and Mussey[2] of Mayo's in 1927 summed up their experimental work on focal infection as a cause of abortion as follows:

> Abortion in guinea pigs has been produced experimentally by the intravenous injection of strains of green producing streptococci isolated from foci of infection in patients who aborted. The premature emptying of the uterus in animals seemed to be due to the elective localization and growth of the streptococci in the placental site and death of the fetus due to invasion of its blood stream by this streptococcus. These streptococci lost their specificity on artificial mediums. Control strains from foci of infection in patients never having aborted injected in like manner and in equivalent doses did not produce experimental abortion.

Talbot[3] in 1922 wrote two very good articles on chronic infection in pregnancy and tried to demonstrate that these foci of infection cause infarcts in the placenta which he, in turn, thought was a factor in producing pregnancy toxemia. Montgomery,[4] however, has recently proved that these infarcts are due to a physiological phenomenon and are found to some degree in every placenta. Talbot cites cases, however, where focal infection was the cause of mastitis and pyelitis and also miscarriage. He even goes so far as to say that probably certain fetal deformities and hemorrhagic disease of the newborn are due to chronic infection in the mother.

* Read before the Evanston Branch of the Chicago Medical Society April 2, 1931.

Johnson, Johnston, and Nicholas[5] of Houston, Texas, make the statement that bacillus coli infection is present in practically every case of toxemia of pregnancy. They think that focal infection lowers the resistance and the bacillus coli have a better chance to invade the organism. They think, too, that focal infection is a factor in the toxemia of pregnancy because bacteria and their enzymes acting on certain amino-acids in the blood stream form poisonous amines. There is no large quantity of amino-acids in the blood stream as a rule but they think that certain things such as renal disease or the ingestion of large amounts of meat might increase them thus causing toxemia.

Doctors W. C. Danforth and B. C. Corbus[6] in a paper on pyelitis in 1927 state that "as pyelitis is secondary in a large group of cases to foci of infection in the teeth, tonsils and sinuses, these regions must be cleared of infection as the first step in its management." Rose and Rollins[7] of St. Louis in a recent article on pyelitis of pregnancy advocate removing all foci of infection as a preventive measure. Focal infection has also been found to be a contributory factor in secondary anemias and I have been able to demonstrate an increase in hemoglobin and red cells following the removal of certain foci of infection even in pregnancy and a few cases of anemia that have persisted long after delivery have been benefited by the removal of certain chronic infections.

The cervix is not thought of as a common source of infection but it has been definitely proved to be in a small number of cases and although conception is not apt to take place if the cervix is badly infected, nevertheless now and then it does occur. Mayo,[8] Langstroth, Sturmdorf, Rosenow and Dickinson have all cited cases where the cervix proved to be a focus of infection capable of causing systemic symptoms. If the patient should present herself prior to conception for a check-up, as she many times does, the cervix should be treated and put into the very best condition possible because after pregnancy begins the secretions coming from the cervix may furnish a great deal of discomfort and may in some cases, I think, even lead to miscarriage.

These focal infections are found most commonly in teeth, tonsils, sinuses and the kidney. Of course, the most common location is the teeth or tonsils and especially the teeth for an area of infection at the base of a tooth can drain only into the circulation. It may produce no symptoms whatever but it is constantly exposed to pressure by mastication. That the kidney is a focus of infection is probably not entirely correct but we do see a number of cases that have a chronic infection in one or the other kidney that will respond to proper drainage or treatment and these chronically infected kidneys should be treated prior to conception or at least in the early months of pregnancy.

In November, 1929, I decided to take a full mouth *x*-ray on all patients coming for the care of pregnancy. Since that time 242 pregnant women have come for care. The *x*-rays of their teeth have all been taken by Dr. Melvin A. Root, Jr. The patient is given a slip to Dr. Root who in turn sends me a written report on the condition of her teeth. The *x*-ray is then mailed to her dentist within a few days. If Dr. Root's report shows the presence of infection, I communicate with her dentist and in some cases examine the film also. If the three of us agree that an apical abscess exists, she is advised to have the tooth removed. Out of these 242 women, 57 reported at the time of their first visit that they had had a recent full mouth *x*-ray, that is, within a few months. This, I believe, goes to show that they are a class of women accustomed to very good dental care. One hundred and eighty-one full mouth *x*-rays were taken and 4 women have not yet submitted to the examination. Of these 242 patients, 36 had apical abscesses which represents 15 per cent. In other words, from routine examination of the teeth of patients in this com-

munity coming for the care of pregnancy, we are able to demonstrate that 15 per cent are harboring chronic infection. Besides these infections we would naturally expect to find other sources of infection that the *x*-ray is unable to show, such as tonsils, and such is the case for in 1929 I had 9 cases tonsillectomized and last year, 3 cases. Out of the 36 cases of apical infection discovered during this period of seventeen months, 25 patients had extractions performed, 1 had a tooth drained, 4 refused and 6 are yet to have it done. These six women have signified a willingness to do so, however, at their earliest convenience.

Some writers state that they do not think dental extractions and other dental work should be done because of the possibility of dissemination of infection causing miscarriage, etc.; but I feel sure that the patient runs a greater risk by harboring the infection than she does by having it removed. None of the 25 patients having extractions miscarried and none of the patients that I have had tonsillectomized have miscarried.

It is very hard to demonstrate any benefit from a preventive procedure such as the removal of various foci of infection but since adopting the measure as a routine, I have had fewer cases of pyelitis, mastitis and toxemia.

SUMMARY

In summarizing the preceding I wish to state the following:

1. It is probable that about 15 per cent of all women presenting themselves for the care of pregnancy in this community are harboring focal infection at the apices of their teeth.

2. Since focal infection has been very clearly demonstrated to be a source of danger to the average pregnant woman, I think that in the routine care of pregnancy we should include a full mouth *x*-ray of the patient's teeth as a part of the first examination.

3. Since harboring a chronic focus of infection is probably a greater risk than its removal, I think all foci of infection should be removed early in pregnancy.

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DIAGNOSTIC PNEUMOTHORAX

A GUIDE IN THE LOCALIZATION, TREATMENT, AND PROGNOSIS OF PULMONARY ABSCESSES*

WILLIAM B. FAULKNER, JR., M.D.

SAN FRANCISCO, CALIF.

THERE are certain underlying mechanical differences caused by pleural adhesions even in those patients with pulmonary abscesses who present strikingly similar histories, physical findings, and roentgen-ray shadows. These adhesions can be visualized and their extent, localization, and influence can be measured if air is introduced into the pleural cavity. Diagnostic pneumothorax aids not only in selecting the proper type of treatment and in guiding the prognosis but also in presenting the problems to be met at operation so that one may be prepared for them. Pneumothorax is easy to perform and does not aggravate the patient's condition.

METHOD

The air is introduced under local anesthesia by a method similar to that used for the induction of a therapeutic pneumothorax in pulmonary tuberculosis. The patient is placed in a horizontal position with the side uppermost into which the air is to be introduced. This is a precautionary measure against the possibility of a cerebral air embolus (Fig. 1). An attempt is made to introduce the air into the pleural cavity at a point quite distant from the diseased are so as to lessen the chances of perforating the abscess and soiling the pleura. We have not had a case of empyema directly attributable to a diagnostic pneumothorax.

AMOUNT OF AIR

The amount of air used is governed by the intrapleural pressure, the vital capacity, and the subjective reaction to the air.

Intrapleural Pressure. Intrapleural pressure readings must be taken on these patients during the diagnostic pneumothorax to prevent, as far as possible, the danger of tearing adhesions and rupturing the abscess into the pleural cavity. The manometer readings keep the surgeon informed as to the intrapleural pressure and serve as a guide to the amount of air that should be used. Whenever a negative intrapleural pressure is shown, air can be introduced with safety. Pressure readings should be rechecked after the introduction of each 25 to 50 c.c. of air, and if the pressures remain negative it is safe to continue. Usually 250 to 300 c.c. of air will be sufficient to give the desired information, but sometimes smaller or slightly larger amounts are required.

There will be no manometer readings if the pleural cavity is completely obliterated by adhesions, and in such instances no air should be introduced.

Vital Capacity. The estimation of the vital capacity is important especially in patients with low vitality. Air must be introduced with caution and in small amounts when the vital capacity is diminished and it should be rechecked several times during the introduction.

Subjective Reaction to the Air. If the patient experiences extreme pain, restlessness, giddiness, shortness of breath, or any other unusual sensation during the induction of the pneumothorax, the procedure should be discontinued immediately regardless of the amount of air that has been introduced.

X-RAY AND FLUOROSCOPY

The needle and cannula are left in place during the fluoroscopic examination and while the roentgen plates are being taken.

* Read before the Surgical Section, California State Medical Society, May, 1930.

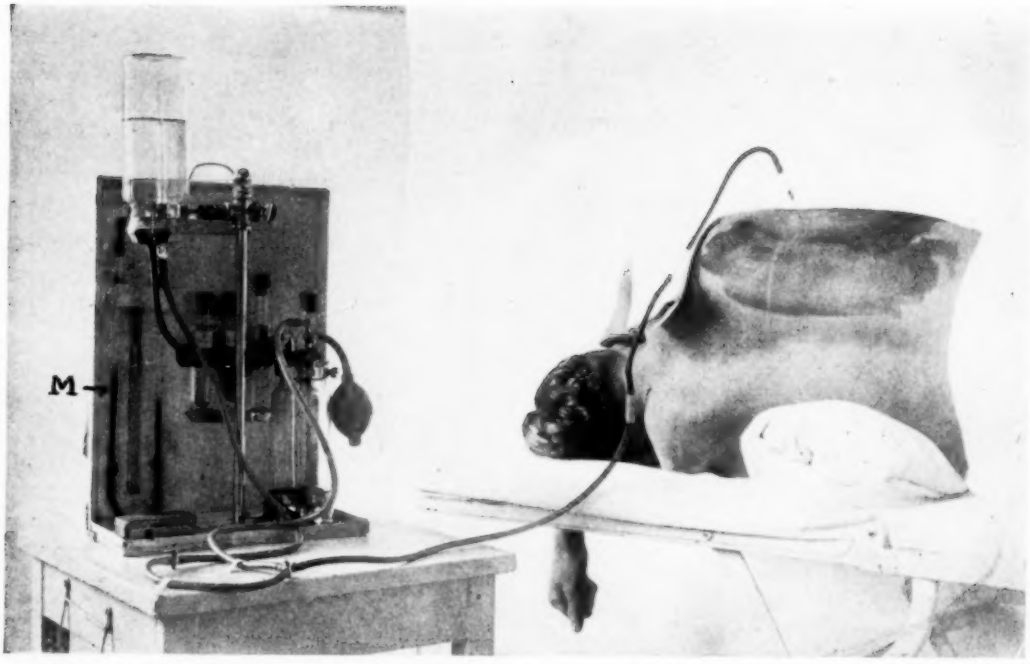

FIG. 1. Position of patient during induction of diagnostic pneumothorax. M, manometer for registration of intrapleural pressures. (We have found the Singer-Phillips pneumothorax machine quite satisfactory for this work.)

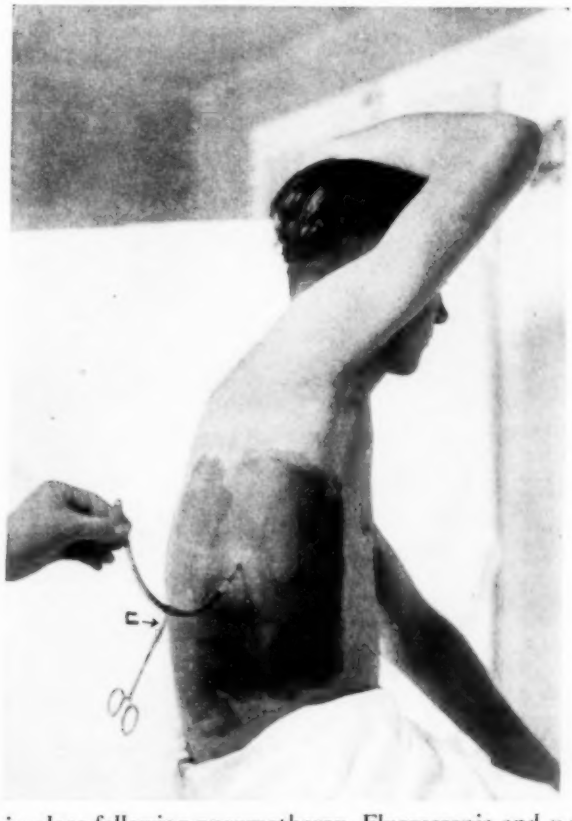

FIG. 2. Needle and cannula in place following pneumothorax. Fluoroscopic and *x*-ray studies are to be made. C, clamp to prevent escape of air from pleural cavity.

More air may be introduced if necessary and withdrawn at the end of the procedure (Fig. 2).

2. Conservative measures with supplementary minor surgical procedures: avulsion of the phrenic nerve.

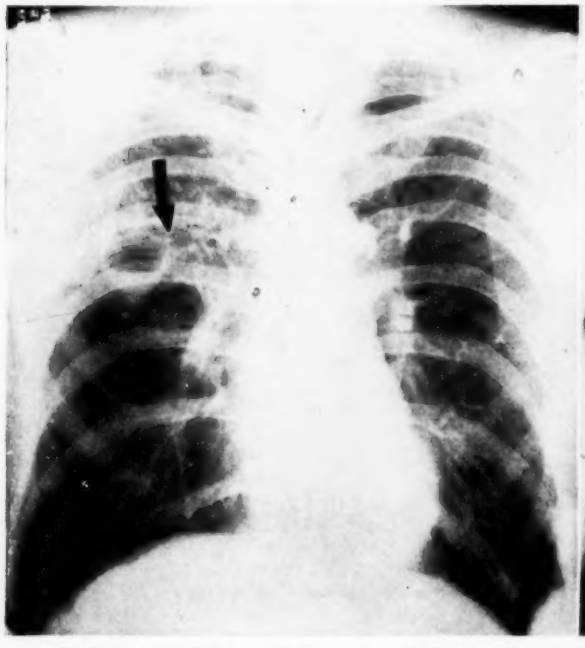

Fig. 3. Pulmonary abscess of three months' duration. Arrow indicates mouth of bronchus leading to abscess.

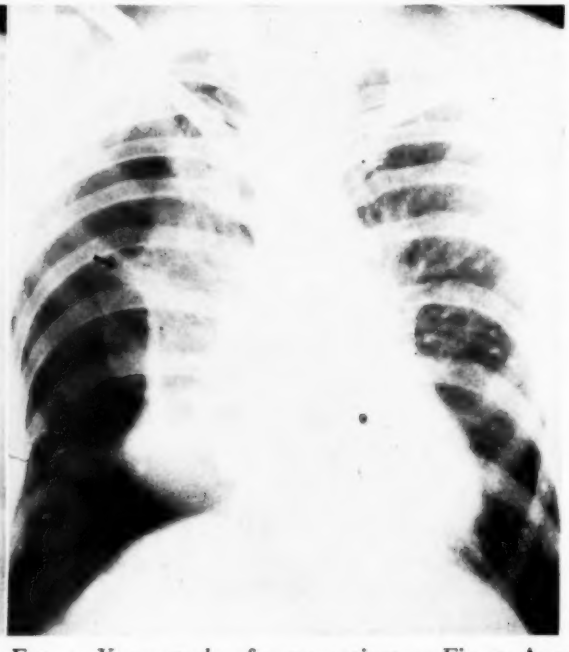

Fig. 4. *X*-ray study of same patient as Fig. 3. Arrow indicates site of former abscess. Air in pleural cavity demonstrates that the abscess is not adherent to lateral chest wall. Abscess has been compressed and collapsed by pneumothorax. Pneumothorax therefore continued as treatment.

It is advisable to make several roentgenograms; usually three are made in these positions: erect postero-anterior, lateral, and horizontal. The horizontal film is exposed while the patient lies with the affected side uppermost; the film is held against the anterior chest wall while the *x*-ray tube is placed posteriorly. If the examinations suggest a posterior abscess, the anteroposterior projection is employed to give better definition and more accurate localization of the site and extent of the abscess.

CLINICAL APPLICATION

Diagnostic pneumothorax has enabled us to segregate lung abscess cases into three main groups which respond to the following treatments:

1. Conservative measures: postural drainage, bronchoscopy, and pneumothorax.

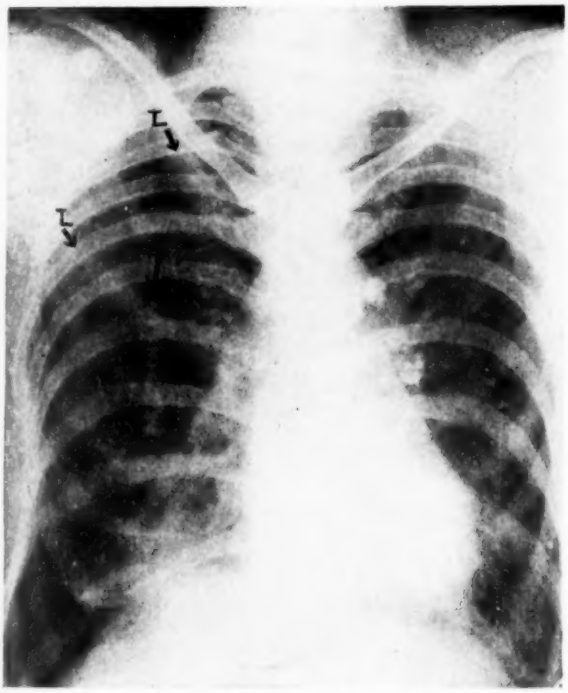

Fig. 5. *X*-ray study of same patient as Fig. 3. Lung is now reexpanding and patient is free from all symptoms. s, site of former abscess. l, lung edge.

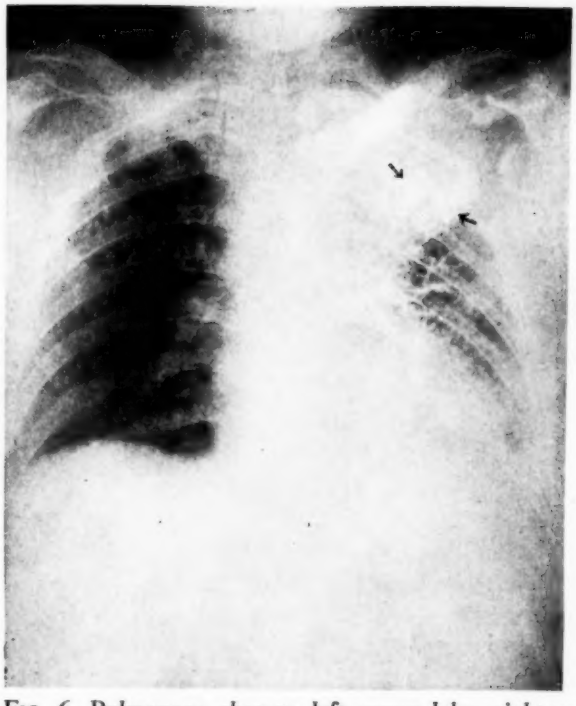

FIG. 6. Pulmonary abscess, left upper lobe, eighteen months' duration. Arrows indicate multiple abscess cavities filled with iodized oil (campiodol).

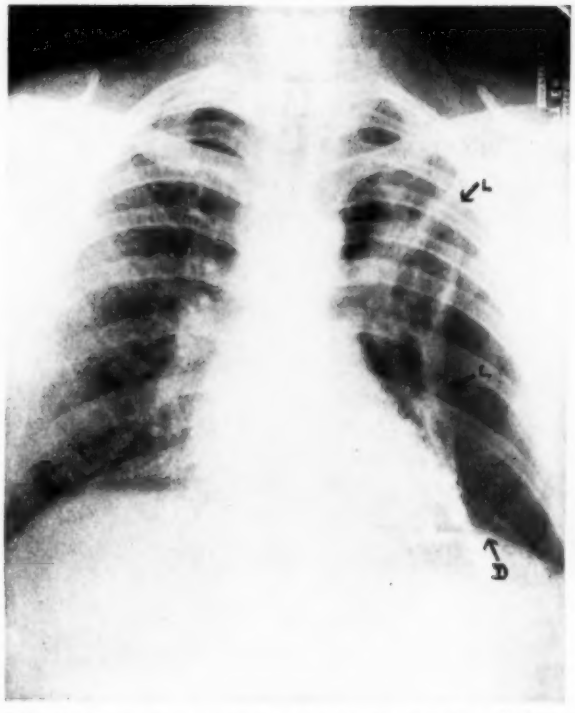

FIG. 7. *X*-ray study of same patient as in Fig. 6. Lung edge (L) is adherent to upper lateral chest wall and to diaphragm (D). Inspiratory diaphragmatic movement tugs on abscess and prevents healing. Left phrenic nerve avulsion is indicated to prevent this unfavorable diaphragmatic action, and to permit lung compression and healing of abscess.

FIG. 8. Diagnostic pneumothorax shows that lung is adherent to anterior and posterior portion of diaphragm as well as to upper chest wall. These adhesions hold lung stretched out like a tent and consequently abscess cannot heal. L, lung edge.

FIG. 9. Roentgenogram of same patient as Fig. 8. Phrenic nerve has been avulsed, diaphragm has ascended into thoracic cavity and lung has been compressed.

3. Major surgical procedures: open drainage, cautery-pneumonectomy, partial thoracoplasty, or lobectomy.

Fig. 10. Left-sided peripheral pulmonary abscess. Iodized oil did not enter abscess. Pneumothorax is to be done to determine presence or absence of adhesions.

If diagnostic pneumothorax demonstrates a pulmonary abscess that is not adherent, a cure may be effected by the combined use of postural drainage, bronchoscopy, and pneumothorax treatments (Figs. 3, 4, 5). The bronchoscopic treatments accomplish and maintain adequate drainage while the pneumothorax evacuates pus from the diseased lung and affords sufficient lung compression and pulmonary rest to permit healing. In these cases pneumothorax has served not only as a diagnostic measure but also as a guide in therapy and as an indication of the probable outcome. Diagnostic pneumothorax should not be used if there is any indication that the abscess has a thin wall. Whenever the bronchus leading to the abscess is occluded, the diagnostic pneumothorax should always be preceded by a bronchoscopic treatment and aspiration of the occluding material.

Fig. 11. Roentgenogram of same patient as Fig. 10 after introduction of air into pleural cavity. Film was exposed with patient lying on his right side. Pneumothorax demonstrates adhesions to overlying lateral chest wall (c-c). Lung is also adherent to medial half of diaphragm (d) but is free elsewhere. Open surgical drainage of abscess is warranted and can be done by a single stage operation because of adhesions. Ribs to be resected clearly indicated (two ribs between c-c). Operation confirmed pneumothorax findings.

Should diagnostic pneumothorax reveal adhesions of the diseased lung to the diaphragm, fluoroscopic examination will determine whether the inspiratory diaphragmatic movements cause a tug on the abscess in such a manner as to prevent healing. This unfavorable tug is present most often when the diseased lung is adherent to a point high in the thoracic cavity and also to the diaphragm. If pneumothorax only is continued as a treatment under such circumstances, the patient may be made worse instead of better. Therefore those who fail to improve with the combined pneumothorax and bronchoscopic treatments should have a phrenic nerve avulsion. This avulsion will relax and elevate the diaphragm, compress the abscess, and prevent the undesirable diaphragmatic tug. Therapeutic pneumothorax may be continued with good results after the phrenic avulsion (Figs. 6, 7, 8, 9).

When pneumothorax shows the abscess situated peripherally and adherent to the chest wall, surgical approach is easy and the study of patients with pulmonary abscesses; it is not our intention to minimize their value.

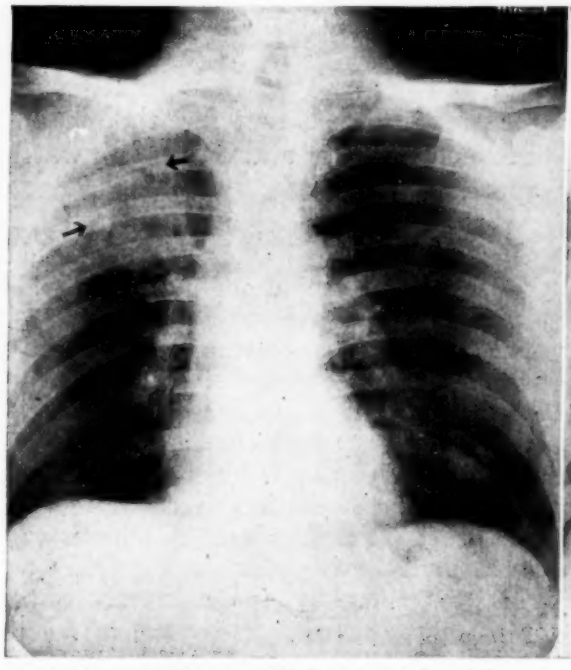

Fig. 12. Pulmonary abscess of right upper lobe, eighteen months' duration. Examinations negative for tuberculosis.

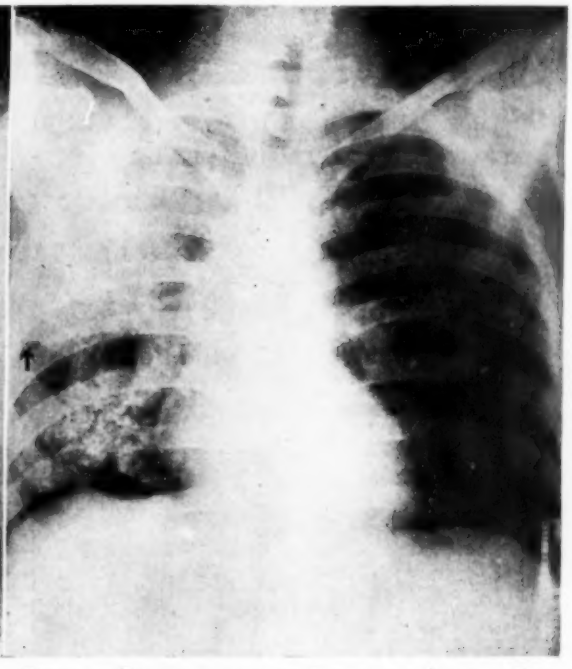

Fig. 13. Roentgenogram of same patient as Fig. 12. Diagnostic pneumothorax shows that pleural cavity is almost completely obliterated by adhesions and that diseased area is firmly adherent to chest wall. Arrow indicates small amount of air outlining lower border of upper lobe. Open surgical drainage is indicated. Operation confirmed diagnostic pneumothorax findings.

safe and the patient may be offered prospects of a cure by open drainage (Figs. 10, 11).

Patients with chronic lung abscesses, in whom attempted pneumothoraces have demonstrated a complete obliteration of the pleural cavity, are unlikely to be cured by conservative measures. In this group surgery should be done early because delay may lead to a bronchogenic and metastatic spread of the disease. (Figs. 12, 13.)

COMMENT

Pneumothorax is intended to supplement and not to supplant the other accepted procedures. Frequent examinations, plain roentgenograms, iodized oil injections, and bronchoscopic findings, all are important in

CONCLUSIONS

1. Diagnostic pneumothorax has a wide application in thoracic surgery. It is easy to perform and in properly selected cases is rarely followed by complications.

2. The use of this procedure permits early selection of cases that probably will require surgery and allows us to recognize those that likely will get well by conservative measures.

3. The employment of pneumothorax along with bronchoscopy and iodized oil has increased our percentage of cures, has shortened the time of recovery, and has avoided operation in a number of cases.

UNILATERAL IDIOPATHIC HYPERTROPHY OF THE MALE BREAST*

FREDERICK CHRISTOPHER, M.D., F.A.C.S.

WINNETKA, ILL.

BAILEY[1] states that up to 1924 but 150 cases of unilateral idiopathic hypertrophy of the breast were on record. The present case is therefore thought to be of sufficient rarity and interest to warrant report.

FIG. 1.

Patient I. T., aged sixteen, was admitted to the Evanston Hospital on June 11, 1930. The history was negative save for the fact that since about one year previously the patient had been conscious of progressive enlargement of the right breast. When in a swimming suit or engaged in heavy work on shipboard in a track suit, his condition caused him much embarrassment because of the jeering comments of his associates. The physical examination was essentially negative save for the right breast. This was markedly and uniformly enlarged so as to present the appearance of a female breast. The areola was definitely increased in size (see Fig. 1). The consistency of the breast was that of the mammary gland in the female. The genitalia and hair distribution were normal; the voice was deep; the general physical characteristics were strongly masculine.

Because of the embarrassment to the patient a subcutaneous excision of the breast was decided upon. On June 12, 1930, under avertin and nitrous oxide anesthesia a curved incision was made at the inferior lateral border of the breast. The upper margin of the wound was reflected backward and breast dissected away from the skin and pectoralis major muscle until it was entirely excised. Aside from the necessity of evacuating a serous collection from beneath the skin, the patient made a very satisfactory recovery.

[1] Bailey, H. *Lancet*, 1:1258, 1924.

* Submitted for publication April 30, 1931.

THE EFFECT OF AVERTIN UPON THE NORMAL AND IMPAIRED LIVER*

WESLEY BOURNE, M.D., F.R.C.P., AND B. B. RAGINSKY, M.D., C.M.

MONTREAL, CANADA

AVERTIN, tribromethylalcohol, was first used as a general anesthetic in 1926 in Germany. It immediately claimed extensive attention in that country; the bibliography of the monograph of Anschütz, Specht and Tiemann[1] published last year, covers ten pages. Much for and much against the new compound has been published but there would seem to be little doubt now that the substance has found a definite field of use among the anesthetics.

There is a considerable degree of chemical relationship between chloroform and avertin; the former contains three atoms of chlorine, the latter three atoms of bromine; one is a methane derivative, the other an ethyl alcohol derivative. Because of the halogen content, by analogy with chloroform, it was natural that the question of possible liver damage from avertin arose; chloroform as is well known, is very toxic to this organ. While the question is a legitimate one, investigation alone and not presumption can furnish the answer. One of us in collaboration with M. Bruger and N. B. Dreyer has already published the results of experiments on the effect of avertin upon hepatic function in normal men and dogs.[2] These results indicated that the action of avertin upon the normal liver is practically negligible, about the same as in the case of ether and much less than in the case of chloroform.

It is a matter of importance to know whether an organism with an already subnormal liver withstands avertin as well as a normal one. Increased susceptibility might be due to one or two things. First there is the possibility of greater interference with liver functions than in the normal animal. And second, since avertin is excreted as a conjugated glycuronate, the question of interference with the detoxification of avertin arises. If the conjugation process took place less completely or readily in an organism with a pathological liver, assuming that the process in question occurred in the liver, the action of avertin in other tissues might be more prolonged or more severe. However, Eichholtz[3] has reported that when the liver is injured by ethylene chlorhydrin the detoxification of avertin proceeds normally; this organ would not appear, therefore, to be the only tissue concerned in the conjugation process and any deleterious action of avertin upon it must be confined to interference with other liver functions. So far as these are concerned, we have noted the work of Kaczander[4] only, who could find no evidence of a toxic action upon the liver cells as tested by the effect of the administration of galactose upon the bilirubin content of the blood.

Waters and Muehlberger[5] in their work upon avertin report the histological findings in the livers of three dogs which had received from eight to twenty rectal injections of avertin every two to five days. "The liver parenchyma showed marked swelling of the cells which almost obliterated the capillary field. The individual cells were extremely pale and vacuolated, and showed coagulated, eosin-staining granules throughout. The vacuoles were serous rather than fatty. In one case the cells were distinctly atrophic." On gross examination the liver presented possible fat. The question whether such changes would have been observed following a single administration of avertin is not answered by these observations.

* Submitted for publication September 30, 1931.

Deaths in man following the use of avertin have in some cases been attributed to liver damage. Anschütz, Specht and Tiemann discuss some of these cases in their review and suggest that data similar to those in avertin literature might be obtained if one looked as diligently for liver damage after ether or even local anesthesia.

This communication represents efforts (a) to detect histological changes following avertin in dogs which had had repeated administration of avertin (similar experiments to those of Waters and Muehlberger) and (b) to measure the action of avertin upon the liver function in dogs in which this had been depressed by previously subjecting the animals to chloroform anesthesia.

EXPERIMENTAL

1. *Histological:** Histological examinations of the liver were made in five normal dogs and of the kidneys and heart in three which had previously received from three to six full anesthetic doses of avertin fluid per rectum (0.5 grams of avertin crystals per kilogram of body weight), in the course of one week. Frozen sections were used for the detection of any possible fatty changes.

Of these animals one was in the early stage of pregnancy and another at term, delivering itself of a normal litter of pups during the experiments. It is of interest to note here that the length of anesthesia in these two animals was greatly prolonged, due possibly to the fact that in calculating the dosage of avertin, no allowance was made for the gravid uterus and its contents and to the altered condition of the liver during pregnancy. As will be shown later in this communication, anesthesia is prolonged in animals with damaged livers.

The following is a summary of the histological findings:

Liver: The most consistent change is the presence of swollen parenchymal cells compressing the intercolumnar spaces, with vacuolization of the cytoplasm, (a parenchymatous degeneration compatible with restitution to normal function). The livers of the two pregnant dogs both showed slight fatty changes; in one there was a slight fatty degeneration, in the other a slight fatty infiltration. Of the other three animals, one revealed the presence of a slight fatty degeneration while the remaining two presented no fatty changes at all.

Kidneys: In one instance there was a slight parenchymatous and fatty degeneration; in another, a moderate similar condition and in the third, a dilatation and engorgement of the large vessels.

Heart: Normal in all cases.

These findings confirm those of Waters and Muehlberger[5] Eichholtz states that avertin causes no "Leberverfettung."[6] The histological picture presented here is not at all typical of a chloroform-like action.

2. *Functional:* The effect of avertin upon the liver function was measured by means of the bromsulphalein test according to Rosenthal and White.[7]

As controls, three normal dogs were given 0.5 c.c. of avertin fluid in a 3 per cent aqueous solution per rectum. Twenty-four hours later the liver function was measured by determining the percentage of dye retained in the blood, fifteen minutes after its intravenous injection. In all 3 cases the retention was not more than 5 per cent. Forty-eight hours later there was no fifteen-minute dye retention. Among our previously published results embracing a large series are some showing higher degrees of impairment. The difference, if the present three results represent a difference rather than a fortuitous circumstance, may be due to the fact that in these later experiments the avertin was administered very slowly. Slow administration was found to result in smoother induction with less cyanosis.[8]

Chloroform was then administered by inhalation to seventeen normal dogs in which a control liver function test had been performed; the duration of administration was varied from fifteen minutes to one hour in order to produce different degrees of liver damage, though even in experiments in which the chloroform periods were equal, quite different degrees of functional disturbances may result. The degree of liver damage under these conditions and the curve of restitution to normal function have been worked out previously in this laboratory.[9]

After periods varying from one to six days after the chloroform administration (in order to observe its effect on liver function at differ-

* The authors are indebted to Dr. Bowie of the Department of Histology for the preparation of the sections and to Prof. T. R. Waugh of the Department of Pathology for examining them.

ent stages of liver damage), full anesthetic doses of avertin fluid were administered, and liver function was again determined. The data

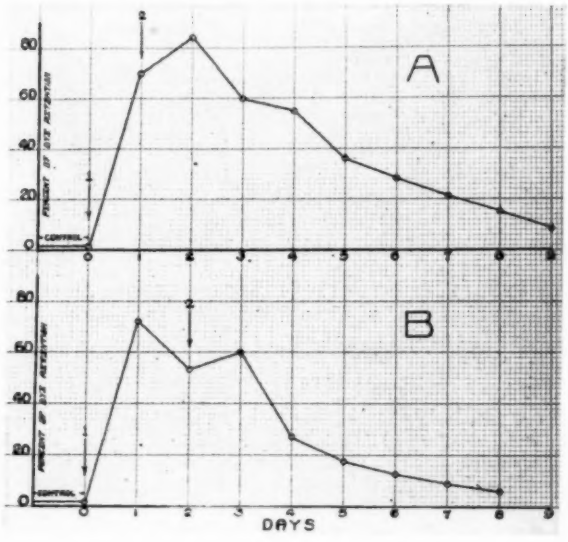

Fig. 1.

also make it possible to observe the effect of avertin on the duration of the liver damage due to chloroform.

Figure 1 (A) represents the composite of three curves obtained by plotting the percentage of dye retention fifteen minutes after its injection in three experiments in which the chloroform had been administered for a half hour and in which the liver damage was relatively severe. At 1 the chloroform was administered (immediately after the control dye test); at 2, twenty-four hours afterwards, avertin fluid 0.5 c.c. per kilogram of bodyweight, was injected rectally (immediately after the second dye test). Curve 1 B is a similar composite of three other experiments in which the avertin was given forty-eight hours after the chloroform administration.

These curves show that the additional impairment of liver function superimposed upon that already present is probably somewhat higher than that produced by avertin in the normal animal (5 per cent as stated previously) but that the return to practically complete normal function is not appreciably prolonged.[9] The average increase in dye retention, or in other words, the decrease in liver function due to the avertin, is from 20 to 30 per cent. In the earlier work referred to here, this

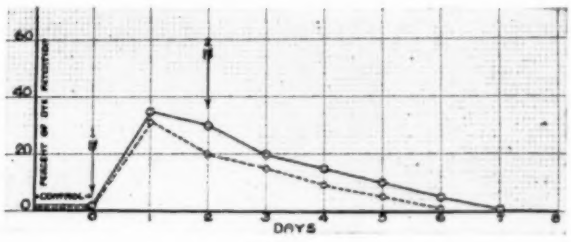

Fig. 2.

degree of dye retention was observed only occasionally.

The effect of avertin on liver function in livers only moderately damaged (fifteen minutes of chloroform) is shown in Figure 2. The broken and unbroken lines represent two separate experiments in which chloroform was administered for fifteen minutes (1), followed in forty-eight hours by avertin (2). It will be seen from this chart that avertin under these conditions has very little effect on liver function. In another experiment (not shown), in which the liver damage had been more severe, the avertin was administered on the *sixth* day following the chloroform (fifteen minute administration). When the avertin was given, the liver function had returned to approximately the same level as in Figure 2 at the second day. The result was identical with those obtained in the other experiments, i.e., there was little if any effect on the liver function or on the curve of recovery.

Figure 3 (A and B) shows the curves obtained when, after severe liver damage had been produced by chloroform inhalation (fifteen minutes) at 1 in the chart, avertin was given at a time when the animals showed all the evidence of delayed chloroform poisoning (four to six days after chloroform administration). In Figure 3 A, even under these circumstances, the deviation from the normal course of recovery is comparatively small and almost imperceptible clinically. This curve is typical of three similar experiments.

In two other experiments of this kind, Figure 3 B, death occurred during the administration of the avertin. Both animals

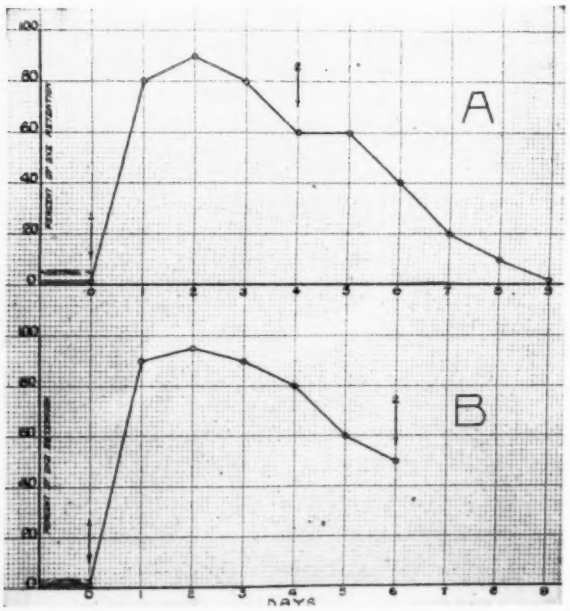

FIG. 3.

were in perceptibly subnormal condition before the avertin was injected. While to avertin must be attributed the immediate cause of death, the fact that death occurred so soon after the avertin was given, diminishes the likelihood of its being due directly to the action of avertin upon the liver. The animals would probably have succumbed to ether or to almost any additional strain because of poor physiological condition generally.

In one experiment in which severe liver damage was caused by the inhalation of chloroform for one hour, the administration of avertin forty-eight hours later produced an increase from 70 per cent to 95 per cent fifteen minute dye retention which lasted for slightly over two days. The liver function returned to normal (no dye retention in fifteen minutes) fourteen days after exposure to chloroform. This is well within the lower limits of recovery for the initial degree of liver damage observed by Rosenthal and one of us.[9]

It has been observed throughout these experiments that with the same dose of avertin, the anesthesia is deeper and lasts longer in dogs with impaired livers than it does in normal animals.[8] It would seem desirable therefore, to reduce the dose of avertin in individuals with impaired livers.

SUMMARY AND CONCLUSIONS

1. Repeated administration of avertin in normal dogs produces only a mild parenchymatous degeneration of the liver and kidneys.

2. Fatty changes in the liver occur occasionally. When they do occur, they are very slight.

3. No histological changes occur in the heart following repeated avertin anesthesia.

4. Avertin produces from 5 to 30 per cent additional damage to liver function; this additional damage varies in proportion to the severity of damage previously produced by chloroform. This added liver damage disappears within twenty-four to forty-eight hours and does not affect the total period of recovery from chloroform poisoning. In the normal animal (not poisoned with chloroform) avertin produces somewhat less impairment of liver function.

5. Avertin has by no means the same damaging effect upon the liver as chloroform.

6. Avertin can probably be used quite safely in individuals with moderate liver damage, though it is perhaps advisable to use a smaller dosage.

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THE CLINICAL MANAGEMENT OF HORSESHOE KIDNEY*

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NEW YORK

Part I†

1. INTRODUCTION

HORSESHOE kidney or fusion of the two kidneys across the midline of the body by an isthmus, is, after double kidney and reduplication of pelves, the most common of all renal anomalies.

The anatomical conception of this congenital malformation has been well established since the early days of observations on the cadaver. Its frequency and the associated pathology as well as the concomitant anomalies and the predisposing urinary stasis and various diseases have also been described by many authors who have written on the subject. Even during the last half century with the beginning of renal surgery, this "rare curiosity" of the ancient days was quite frequently found by the surgeon at operation. Moreover, before a clear-cut clinical diagnosis was ever possible, surgical intervention upon this fused organ was advocated. However, not until comparatively recent years has the surgical importance of this pathological entity been recognized, and it is due mainly to the advantages of the urographic era that accuracy and perfection in the operative diagnosis of the horseshoe kidney disease have become possible.

I shall therefore endeavor briefly to discuss the value of its preoperative diagnosis and its clinical management, and to bring to view what should be the proper treatment of this urological condition.

It is also my purpose to point out the importance of a new clinical entity which, although it has been recently described, has not been given sufficient attention and has apparently not received enough emphasis or recognition, namely, "disease of the horseshoe kidney"

* From the Department of Urology, James Buchanan Brady Foundation of the New York Hospital. Read before the annual meeting of the American Urological Association, New York City, June 12, 1930.

† This monograph will appear in three parts; Parts II and III will be published in the January and February, 1932, issues of The American Journal of Surgery.

or "horseshoe kidney disease," particularly when there is no association of a concomitant disease or any evidence of gross pathology present in the fused organ. When the horseshoe syndrome of clinical manifestations is due to the prominent position across the midline which the anomalous kidney occupies—pressing upon the great abdominal vessels, the aorta and vena cava, the thoracic duct, the solar plexus with its splanchnic, pneumogastric and phrenic nerves, the lumboaortic plexus and the sympathetic and parasympathetic nerves which run alongside the midline—this pressure constitutes a sufficient cause for the long-standing complaint, which is expressed clinically in intermittent attacks of indefinite abdominal pain referred to the epigastrium or the umbilical region, or in pain across the lower back, commonly accompanied by marked chronic constipation and sometimes by gastrointestinal disorders, and later by urinary disturbances, for which the patients are referred to the urologist for examination.

Furthermore, it is an entity which deserves painstaking attention with regard not only to the diagnosis, which is nowadays urologically well recognized, but also to the method of treatment, which obviously should be, first, medical, second, urological, and third, surgical. I wish here to stress the surgical aspect, because in many instances no permanent cure or relief of symptoms can be obtained without the surgical division of the isthmus by a symphysiotomy procedure, such as was described by Rovsing in 1911, and later practiced and advocated by Papin, Martinow, Kroisz, Van Houtum and Frank Kidd as the most logical and as the anatomo-physiological method, relieving the pain and pressure caused by the constant tension of the renal isthmus upon the organs of the blood, nerve and lymphatic supply which run perpendicularly alongside the lumbar column and upon which the undivided isthmus of the horseshoe kidney makes pressure by its weight.

Although it appears that normal horseshoe kidneys do exist and are found at autopsy or in the dissecting room, we cannot overlook the fact that potentially all individuals who have been born with horseshoe kidney and who come to us for examination come because they are suffering with some chronic renal disease due mainly to the unusual and abnormal position of the fused organ and its excretory apparatus.

Many operations upon the horseshoe kidney have been practiced in America as well as abroad, but so far as the records and literature are concerned I have not been able to find a single instance in which this conservative procedure has ever been performed on this side of the world. I hope, therefore, that this present study will serve to stimulate attention to the consideration and discussion of the value and also the convenience of this method of treatment in horseshoe kidney disease.

It has been my good fortune, after the study of my own personal observations, to review the cases seen in the last ten years at the Urological Department of the James Buchanan Brady Foundation of the New York Hospital where 25 cases of horseshoe kidney have been observed. Of this series of cases which I am reporting, 4 were found at post mortem, 2 were diagnosed at operation and 19 were diagnosed urologically and urographically previous to operation.

All of the 25 clinical observations have revealed much unknown and unsuspected pathology and have been of great clinical value as the basis of this study.

I am indebted to Dr. O. S. Lowsley, the Director of the Urological Department of the New York Hospital, and to the members of the staff of that institution for having permitted me to use their cases for this presentation.

II. Classification

From the anatomical standpoint as well as from that of the pathological and clinical management, it is important to establish at the outset a classification that may serve to comprise not only the different types and forms of this anomaly but also to summarize more clearly and comprehensively the different aspects of the clinical conception of the horseshoe kidney.

In Figures 2 and 3 I have tabulated in a graphic manner the different types of this renal anomaly as collected from the various authors who have written on this subject.

But horseshoe kidney classically must be divided, for its better clinical and surgical management, as Gerard has proposed, into two essential groups, namely, the symmetric and the asymmetric. The first group comprises 2 types: (1) those cases in which the fusion of

the two organs is made by the lower pole of the two kidneys forming a semicircular bridge of renal parenchyma with the concavity above, and (2) those in which the fusion is made by the union of the upper

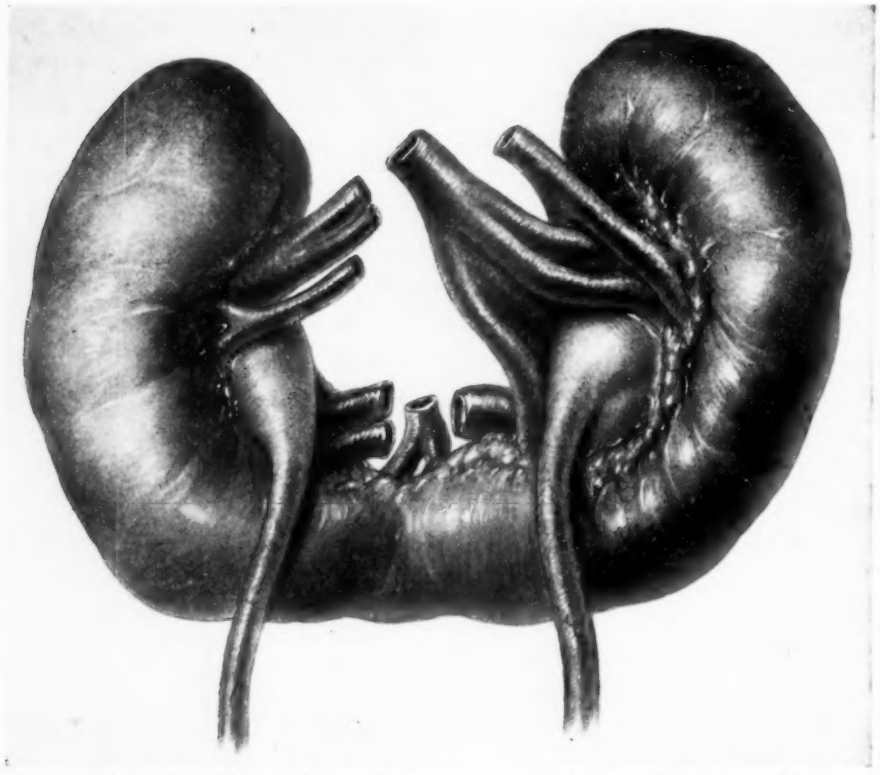

FIG. 1. Horseshoe kidney drawn from autopsy specimen of Case XXII. Notice the extreme multiplicity of the blood supply and the typical ventral position of the pelves and ureters, as is the rule in the symmetric type of horseshoe kidney disease.

pole of the kidneys, with the concavity below. In both instances the fused organ sits, like a body on horseback, on the spinal column at about the level of the third and fourth lumbar vertebrae, with an upper or lower angle in the shape of a U or V, with all the characteristics of a horseshoe, from which this rare anomaly takes its name. As a rule the isthmus is of true renal parenchyma and is seldom a band of fibrous tissue. The pelvis of the kidney is anterior to the blood supply of the organ, and the ureters run in front and at times perpendicular to the isthmus of the fused organ, and therefore parallel to the vertebral column and median line (see Figs. 1 and 2).

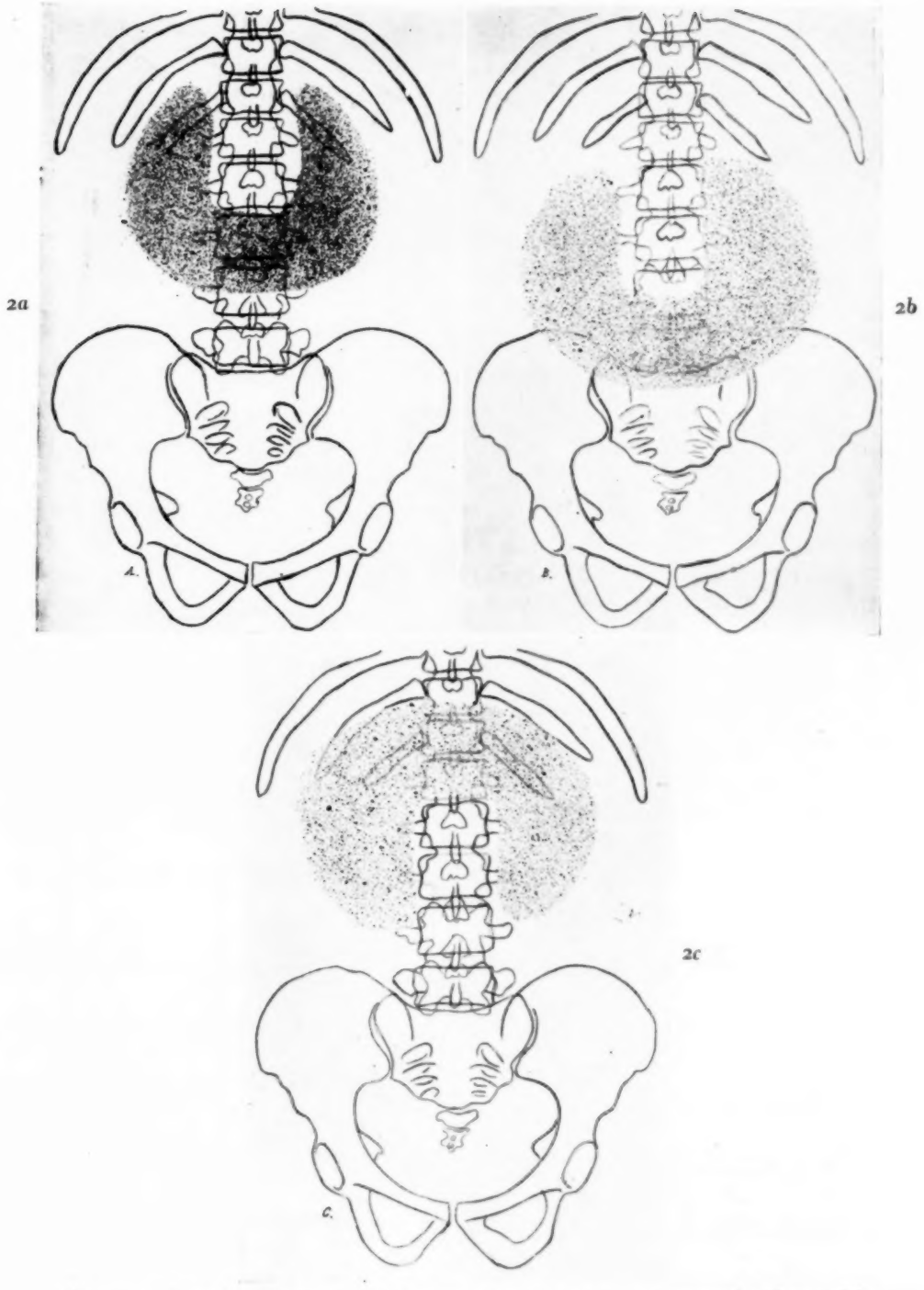

FIG. 2, *a*, *b*, and *c* illustrate the forms and positions most commonly observed in horseshoe kidney disease. *a* and *b* represent the two most common types and positions of the symmetric horseshoe kidney as found in this series of cases, while *c* is the more rare instance, in which fusion of the two halves occurs by the upper pole with the concavity facing downward as in Case XXV.

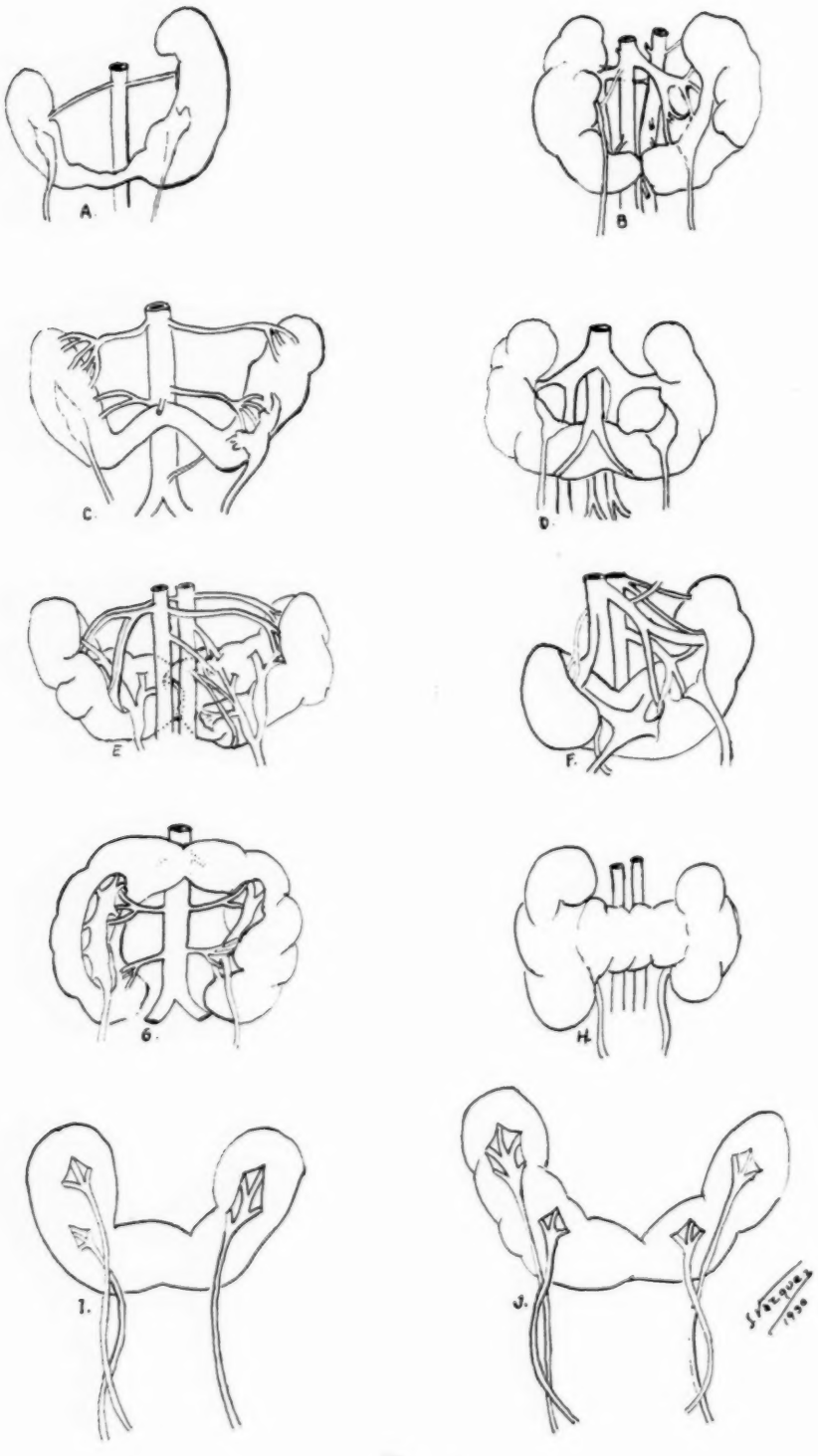

FIG. 3.

This is the true type of horseshoe kidney and the one more commonly known, which I shall discuss in this presentation. In this series of 25 cases of the symmetric or uniform type, the fusion in 24 was made by the lower pole, consequently with the concavity facing upward, while in only one of the cases did the fusion prove to be at the upper pole, representing quite a rare instance, which was found in the specimen removed at post mortem (see Fig. 5).

The second or asymmetric type of fused kidney, where the fusion of the two organs adopts an anatomically different form, with regard to shape, location and position, is more rarely seen and must of necessity be described under special types of renal fusion. According to the form they adopt they are called unilateral fused kidney, L-shaped renal fusion, fusion en galette or disk form, sigmoid fusion, fusion en masse and fusion without form, all of these rare types being generally unions of ectopic kidneys, placed low in the bony pelvis or located at one or either side of the vertebral column. These varieties of fused kidney and crossed ectopic kidneys deserve painstaking attention, and I will reserve discussion of them for another communication on asymmetric renal fusion.

Undoubtedly the frequency with which horseshoe kidney is diagnosed in vivo will markedly increase as time goes on, due mainly to the urographic era of diagnosis. Before the era of pyelography the rate in autopsy findings was about 1 in 1000, while in the last

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FIG. 3. Represents the different types of horseshoe kidney as they have been collected from the literature.

A. Fusion of the two lower poles by a thin membranous tissue running across the midline.

B. The fusion of the two organs is by contiguity of the two lower poles by a fibrous tissue situated in front of the aorta and vena cava.

C. The union of the two organs is made by a narrow band of parenchymal renal substance constituting the isthmus of the horseshoe kidney.

D. The fusion of the two organs is made by a true gross isthmus of renal parenchyma which appears to be the most common type on record and the one most frequently observed clinically.

E. Shows the very rare instance in which the isthmus of the fused organ lies behind the great abdominal vessels.

F. The fusion is of the asymmetric type where one-half of the fused organ is larger than the other, and one pelvis lies in the middle portion of the renal isthmus.

G. The isthmus of the fused organ is formed by the union of the two upper poles with the concavity facing downward.

H. The fusion of the two organs is made by a band of renal substance at the middle portion of the organ, forming the shape of a letter H.

I. Represents the typical symmetric type of horseshoe kidney with unilateral duplication of pelves and ureters.

J. Horseshoe kidney with bilateral duplication of pelves and ureters.

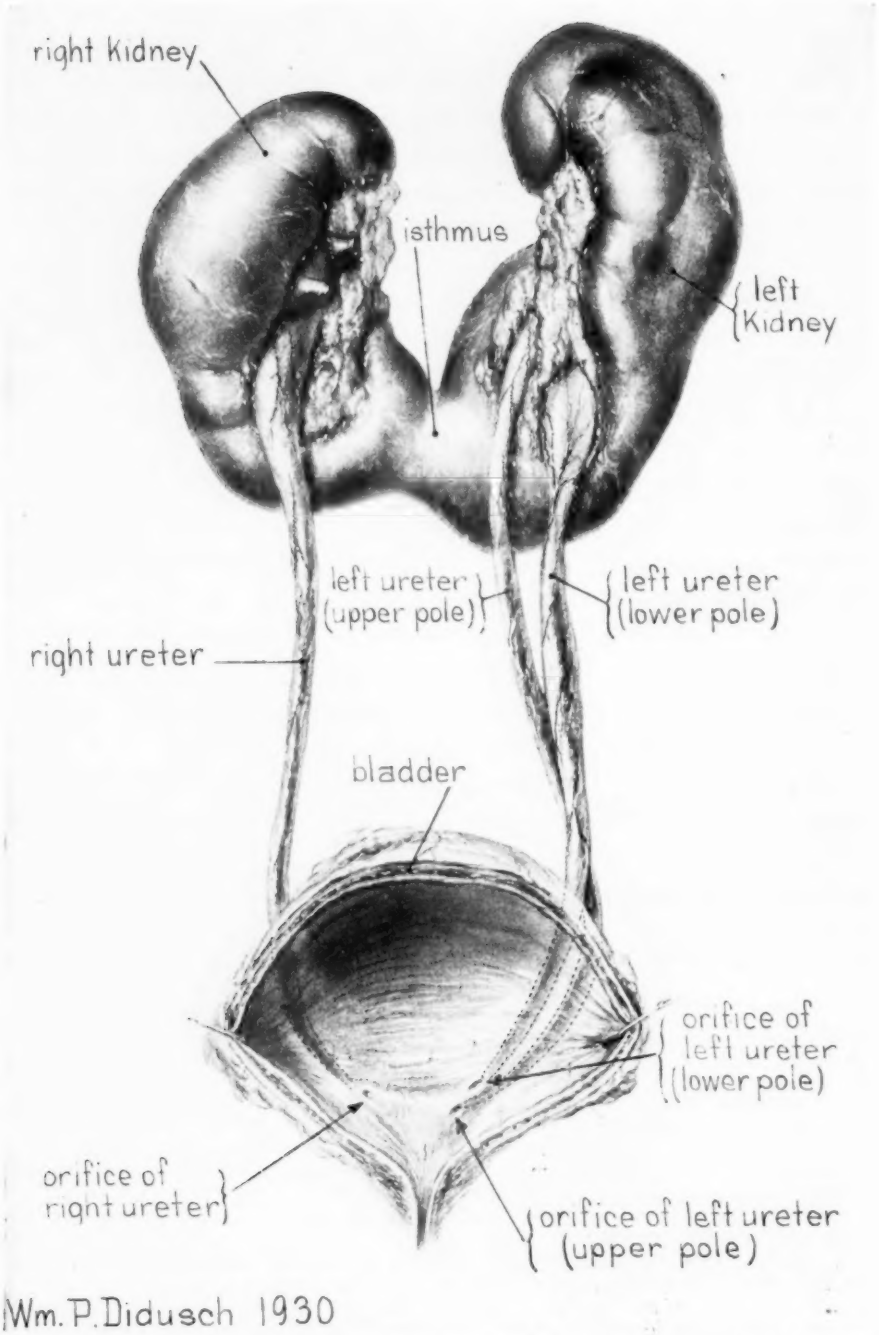

FIG. 4. Drawing from post-mortem specimen of Case XXIV representing a horseshoe kidney with fetal lobulations of the anterior surface, and with double pelves and ureters on the left side, opening normally into the urinary bladder. This patient was a man thirty-five years of age and the autopsy disclosed a retroperitoneal lymphosarcoma involving the anatomic structure surrounding the horseshoe kidney.

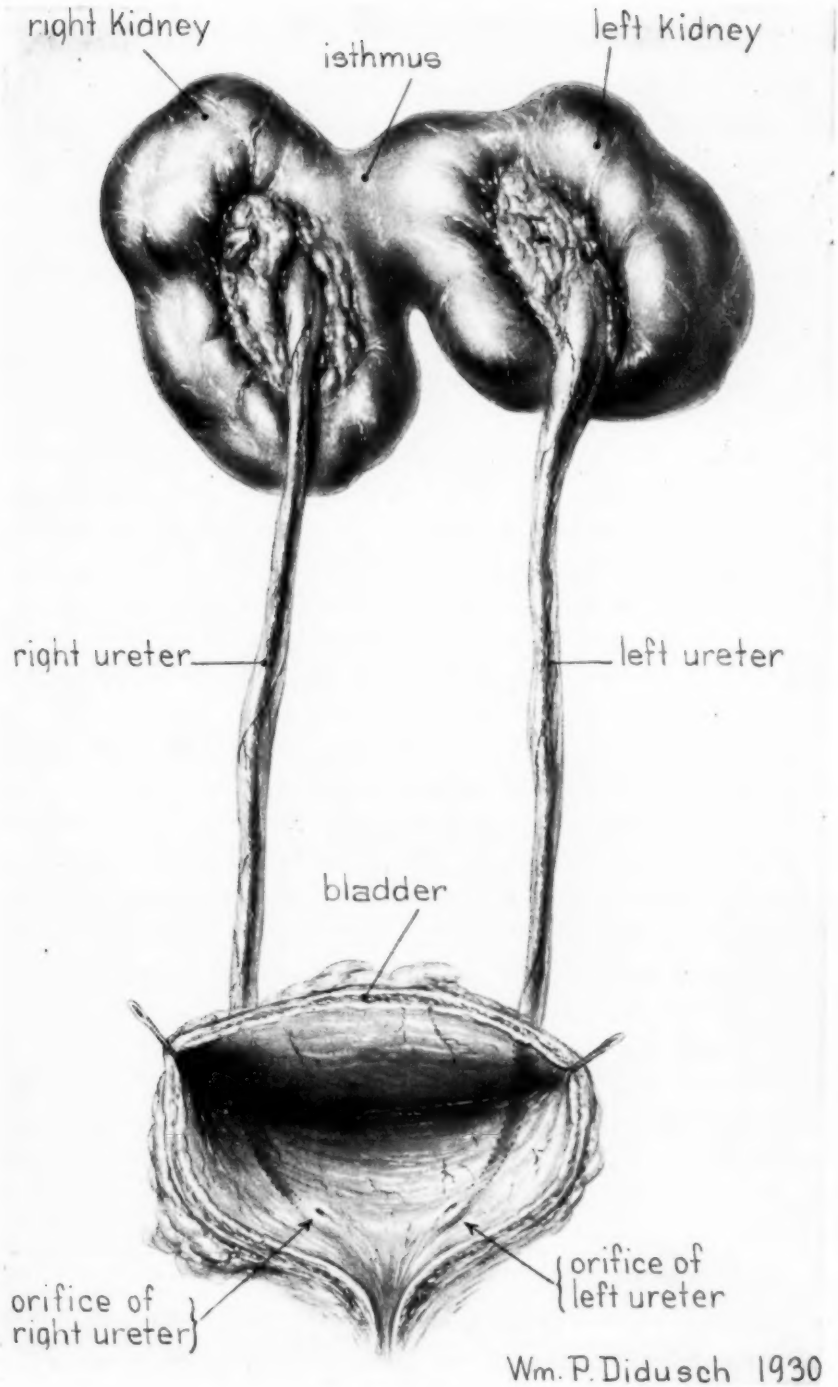
right Kidney
isthmus
left Kidney
right ureter
left ureter
bladder
orifice of right ureter
orifice of left ureter
Wm. P. Didusch 1930

FIG. 5.

two decades with systematic urographic examination it has increased in vivo to about 1 in 400. Hence it is very important to classify these renal anomalies finally into two great categories, namely the normal horseshoe kidney with clinical symptoms and the pathological horseshoe kidney with associated lesions, both of which clinical entities I shall describe collectively as the horseshoe kidney disease.

III. History and Literature

The history of the horseshoe kidney is as ancient as creation itself, since it has its very origin in an embryological congenital malformation found from the earliest days of medicine and anatomical sections in cadavers. The literature of this subject is so extensive and the names of authors who have written on it are so numerous, that it is beyond the scope of this presentation to discuss them in detail or even to mention all of them by name.

However, in modern days of medical science, marked especially by the continuous progress made, it is possible to divide the historical aspect of this study into three distinct eras.

The first era comprises the early days of medicine and cadaveric observation when horseshoe kidneys were found only at post mortem, and the malformation was regarded merely as a "rare anatomical curiosity" without clinical or true pathological significance. Among the first to mention such cases was Berengari da Carpi in the year 1522, and later we find in this connection the names of Morgagni, Cruveilhier, Bartholin, Bellini and other early anatomists. But it is to Morgagni that the credit belongs for the first anatomical description of horseshoe kidney, as well as for the physiological observation of the associated pathology often resulting from the lack of mobility and from the pressure which the anomalous viscus exerts upon the organs of the circulation. Rayer also wrote upon the subject and illustrated it well in his remarkable atlas published in 1837 in which he also called attention to the degree of fixation and compres-

Fig. 5. Drawing from autopsy specimen of Case xxv, representing the very rare instance of renal fusion made near the upper pole by a true isthmus of renal parenchyma, lying in front of and across the great abdominal vessels. The pelves are of intrarenal type and are anteriorly situated. The ureters, therefore, run down from the upper portion of the organ and interfere with the physiologic dynamic function of the excretory apparatus. The two ureters open normally into the bladder. Patient was a woman of fifty and while the direct cause of death was undetermined, it appears that the pressure, tension and fixation of the organ upon the great vessels produced an aneurysm of the arch of the aorta and hence were possibly the direct cause of death.

sion of the renal mass upon the abdominal vessels, producing in some instances aortic aneurysm, cardiac hypertrophy, thrombosis of the iliac veins, and sometimes causing ascites and edema of the extremities for lack of circulation. In those early days all the observations were drawn from autopsy specimens and studies on the cadaver. In fact all cases were considered as rare curiosities or anatomical monstrosities, with the most mysterious and enigmatic elements predominating in the situation.

The second period dates from the beginning of renal surgery, about half a century ago, when horseshoe kidneys were accidently discovered and diagnosed at operation during an exploratory laparotomy procedure for an undetermined abdominal tumor. In such cases the surgeon had to close and finish the operation without the accomplishment of any beneficial result to the patient, and without any surgical attempt upon the anomalous fused organ being ever considered feasible, owing mainly to lack of diagnosis and proper technique and to the potential risk of undertaking a surgical impossibility. Moreover, during this period much confusion existed, due mainly to the unclassified types of cases reported in the literature and to errors and misleading diagnoses, certain authors having mentioned a few instances in which a fused ectopic unilateral kidney was mistaken for an abdominal tumor and removed, with consequent anuria and death.

The third period may be divided into two essential parts. First, we have the era of clinical diagnosis, when operations upon the horseshoe kidney were performed. In thin patients the diagnosis was made on physical examination by deep palpation of the isthmus of the horseshoe organ across the midline of the vertebral column, with due consideration of the clinical manifestation and the concomitant pathology present in the fused kidney. Among the first successful surgical cases reported are those of Israel, Rumpel, Kuster, Newman, Kumwell, Rovsing and Albarran, when various operations, including nephrolithotomy, pyelolithotomy and heminephrectomy on the horseshoe kidney, appeared to be successfully performed. Secondly, we find the era of accuracy in diagnosis, starting at the beginning of the 20th century with the introduction of the roentgen rays and urography, which with the aid of cystoscopy, catheterization of the ureters and renal functional tests inaugurated

the most accurate and brilliant period of the preoperative diagnosis of this most important urological condition.

Hence, the historical aspect can be summarized by saying that the hazardous days of the ancients who, without clinical means of recognition, were confronted by the most enigmatic and difficult situations, have given place to an era of urographic perfection in diagnostic methods, an era which has at the same time introduced new urological treatments and the clear-cut possibility of conservative surgery upon the horseshoe kidney. Among the most important contributions made during the last three decades was that of Israel who, in 1900, was the first to call attention to the possibility of preoperative diagnosis of horseshoe kidney by careful abdominal palpation, provided that the surgeon has in mind such an anomaly. Albarran in those days also successfully performed heminephrectomy and ureteropyeloplasty for hydronephrosis in horseshoe kidney. Martinow on March 9, 1909, was the first to divide the isthmus in order to separate the fused kidney. Rovsing in June 17, 1910, described in full the technique of the new operation for symphysiotomy in horseshoe kidney. He reported a successful case and advocated the use of the method even in the normal horseshoe kidney, recommending the transperitoneal route of approach by the midline of incision as in laparotomy. This operation, however, in view of its many difficulties and lack of proper indications, did not achieve popularity. It presented the disadvantages of uncontrollable bleeding, lack of exposure, danger of secondary infection, urinary fistula and peritonitis, with consequent high mortality. For this reason it has not been performed in European clinics more than a dozen times until within recent years, when Papin introduced a modified technique that made its successful performance possible.

However, before the real value of pyelography was fully recognized and the procedure came into regular vogue, many other contributions of clinical and surgical importance were published. Biron for example, studying the clinical significance of this anomaly in 1909, stated that horseshoe kidney without pathological lesions may be spontaneously painful, owing mainly to lack of mobility and relative fixation. Also Carlier and Gerard, in 1912, reviewing the surgical anatomy of the "rein en fer à cheval" pointed out that the

symptoms and signs in horseshoe kidney are different from the pain in renal lithiasis or ureteral lithiasis. At the same time they quoted the criterion of Morgagni regarding the immobility and the compression exerted by the renal isthmus upon the abdominal vessels, as a causative factor in the pain and lack of circulation, producing in certain instances thrombosis of the iliac veins with edema of the extremities, and sometimes cyanosis and ascites. They also observed that it interfered with the general circulation, causing cardiac hypertrophy and, among the earliest symptoms, tiresome lack of respiration, general anemia, indefinite abdominal pain and marked chronic constipation. Legueu and Papin, in reporting 10 cases found at post mortem, made a survey of the literature from which they collected 212 observations, making a total of 222 cases reviewed. Beyer, in studying 262 cases from the literature, found that the fusion of the upper poles occurred in 18 instances. Botez in 1912 in an exhaustive review of the literature found 5 cases of tumors of horseshoe kidney in children, 4 of which were sarcoma involving one-half of the kidney while the other was a case of carcinoma. He collected from the literature 320 cases, among which he described the following pathological lesions found in horseshoe kidney: 17 cases of lithiasis, 16 of bacillosis, 15 of hydronephrosis, 4 of pyonephrosis, 6 of cyst, 2 of polycystic kidney, 2 of nephritis and 6 of tuberculosis. He concluded that 52 of the patients were incapacitated as a result of the renal condition, and that the ideal treatment in normal horseshoe kidney is the complete division of the isthmus as soon as the diagnosis can be made.

A few other anatomical and clinical contributions to the knowledge of horseshoe kidney were published by Robinson, Newman, Pallazzoli, Papin and Iglesias, Braasch, Rumpel, Löffler, Eisendrath, Zondek and Eggers. Lipshutz and Hoffman, who have tabulated hospital records of various institutions from here and abroad in a total statistical study of 70,502 bodies examined at post mortem, in which 105 horseshoe kidneys were found, estimated a ratio of 1 case in every 671 examined. Legueu and Papin give also approximately the same rate of 1 case in every 500 or 600 individuals. But since the introduction of urography as a routine method of diagnosis, the frequency with which this type of anomaly is discovered in vivo has considerably increased, and horseshoe kidney is quite

commonly found in any active urological service at the present time. Hence the ratio may now be put at about 1 in 100 or 200 pyelograms. Finally, a review of the literature of recent days shows a considerable number of cases reported and operations that have been performed upon the horseshoe kidney. It also demonstrates the exceedingly great clinical and pathologic-anatomic importance of this semicircular mass of renal substance, its enormous frequency and the associated pathology that is commonly attached to it. Such a study reveals also the surgical and clinical aspect of the proper delineation and accurate diagnosis of horseshoe kidney, particularly since the establishment of this enlightened urographic era which has certainly made such diagnosis possible, as has been well brought out in the contributions of Judd, Braasch and Scholl, Rathbun, Eisendrath, Phifer and Culver, Kidd, Colston and Scott, Higgins, Papin, Kretschmer, Young and others.

IV. Embryology

The development of the urogenital system in the human embryo is an exceedingly interesting subject that brings out the complex reasons for the multiple different types of congenital anomalies of the upper urinary tract. Hence, in dealing with a congenital malformation *per se* it is proper to study briefly the genesis of the fused kidney, in an effort to point out the possibility of its early occurrence.

In the embryological evolution of the kidney we are dealing with the development of three essential organs that are formed successively and owe their derivation to both the mesoderm and the entoderm; the last of these three to develop is the one that will serve ultimately to establish the permanent kidney. These three organs are the pronephros, mesonephros and metanephros, respectively. The way in which they develop to form the excretory urinary apparatus has been described and discussed by many authors. It is amazing, however, to observe the rapid evolution of these three distinct organs during the early days of embryonic life when the two renal buds are placed behind the Wolffian duct even before the cloaca is divided to form the rectal and urogenital sinus, and when the kidney anlagen undergo a number of changes, both as to form and position, which undoubtedly are the true underlying factors

in the development of this congenital malformation. Although it appears that the stage of development at which the fusion of the two organs may take place has not been definitely determined, it is

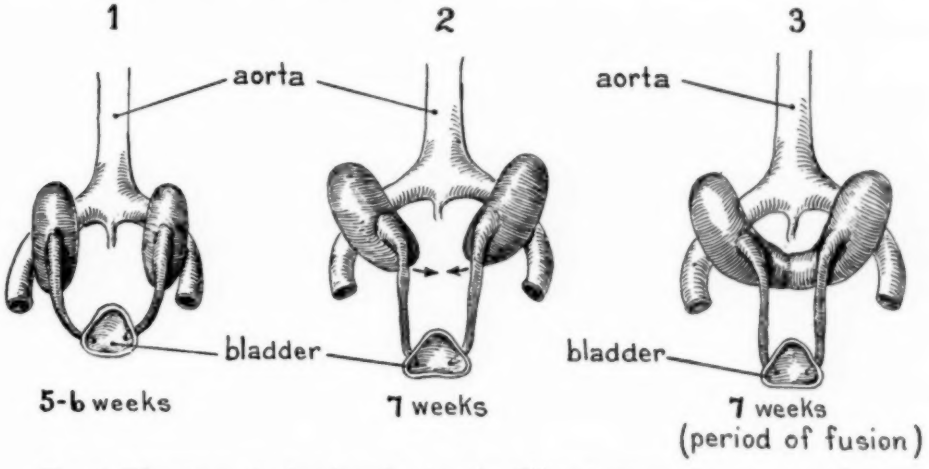

FIG. 6. Diagrammatic drawing showing the different stages of ascent, rotation and migration of the kidneys during embryonic life and revealing the theoretical occurrence of fusion to form the horseshoe kidney.

1. Shows the anterior position of the renal pelvis during the first weeks of fetal life, when the organ is located low down in the pelvis in front of the aorta and behind the future bladder.

2. Represents the two kidneys in their ascent, rotated inward toward the midline, with the two lower poles of the kidney blastemata meeting each other topographically in front of the aorta at the level of its bifurcation into the two iliacs, which may result in a mechanical interference of the normal migration and ascent of the two organs.

3. Illustrates the formation of the actual renal fusion to make the horseshoe kidney during the seventh week of intrauterine life. It appears that this fusion occurs for lack of migration and rotation before the two kidneys ascend from the bony pelvis to their normal position in the lumbar fossae of the adult. The drawing therefore illustrates the possibility of its common occurrence during the early days of embryonic life. Note how the two aberrant pelves remain in front of the renal isthmus. (Modified drawing from Kelly and Burnam.)

nevertheless assumed that the union of the kidney blastema of the right side with that of the left occurs very early in intrauterine life, mainly in view of its topographical relationship and the striking fact that the kidney during its growth migrates and undergoes a rotation around its longitudinal axis. In the course of the evolution of its migration the kidney travels upward or craniad from its original position at the level of the second sacral vertebra to the lumbar region of the adult. The most logical time, therefore, for the fusion of the two organs would appear to be between the fifth and seventh weeks of embryonic life, when the terminal sprouts have

become imbedded in the specialized renal blastema and the two organs are placed closely side by side in the indifferent pre-aortic mesodermal tissue, on the mesial aspect of the caudal poles of the mesonephros (see Fig. 6). Furthermore, it is also possible, as some writers have pointed out, that, as a result of the close relation of the kidneys to the umbilical arteries and to the bifurcation of the aorta into the two iliacs, and the fact that the kidneys are lodged low in the pelvis, the arteries may react as some sort of mechanical obstruction interfering with the upward migration and inward rotation. This would then tend to bring the right and left kidney blastemata together, so that fusion for lack of rotation and ascent may readily take place before the primitive pelvic organ changes its position to become an abdominal organ. If fusion of the upper pole (which is the more rare) occurs, it usually arises earlier than fusion of the lower. At any rate, horseshoe kidneys are generally found to be ectopic, situated low in position about the level of the aortic bifurcation. The renal pelves are always in front, anterior to the blood vessels, and the ureters pass over the ventral surface of the isthmus of the fused organ, revealing the enormous aberration of the anatomical structure from the original position in which it is observed during the early days of fetal life.

Examples of this early renal fusion have been recorded in the literature by Felix, who observed a fused kidney in an embryo 30 mm. in length, or about fifty-five days old, in which the isthmus was caught by the inferior mesenteric artery. Schaeffer also has observed a fusion of the two kidney blastemata in an embryo approximately one hundred days old. Obviously, therefore, the genesis of the fused kidney is best explained on an embryological basis. Hence, one may reasonably conclude that fusion of the two kidney blastemata, to form the two halves of the horseshoe kidney in the adult, in reality occurs much more easily, and its incidence is therefore considerably greater, than the frequency of its clinical discovery has ever led us to suspect.

V. Anatomic Structure

In studying the anatomic structure of the horseshoe kidney, it is necessary on account of its clinical and surgical importance to divide the work as follows:

I. General description of the organ
II. Excretory apparatus, comprising calices, renal pelves and ureters
III. Blood, nerve and lymphatic supply
IV. Topographic relation to the organs situated in front of and behind the semicircular renal substance.

I. The form of the symmetric horseshoe kidney is that of a typical horseshoe or semilunar mass of renal parenchyma. When the fusion is made by the union of the two lower poles, with a concavity looking upward, it constitutes a solid bridge of renal substance extending across the midline of the vertebral column. When the fusion is by the upper poles of the two kidneys, the concavity formed looks downwards. As a rule the form adopted is quite irregular, in the general shape of a V or U, with the point of the angle at the middle portion of the union of the two organs. The two halves seldom have the same form or position; sometimes the right one is larger and lower than the left, or vice versa. The external configuration of the anterior surface of this kidney is very striking, since it retains the marked lobulation and furrows of the fetal organs, while the posterior surface is generally smooth with a middle furrow in the isthmus, formed by the passage of and the close relation to the great abdominal vessels. But what appears to be even more constant and striking is the fact that, in addition to their union, the hilum of the organ is almost always ventrally and inwardly placed as is also the excretory apparatus, which has been definitely shown by embryological and cadaveric observation. These horseshoe organs are situated in front of the great abdominal vessels and behind the peritoneum in the retroperitoneal space.

Two types of horseshoe kidney are encountered: the true, in which the isthmus is an actual solid renal parenchyma, and the false, in which the fusion is made through contiguity of the two organs by a fibrous band of tissue and accordingly without union of the true renal substance. Anatomically as well as physiologically the position occupied by this unique organ in the human body is of paramount importance. Horseshoe kidneys are in general anterior, since their isthmus has become an abdominal organ lying unusually low at the umbilical region and at the level of the fourth or fifth lum-

bar vertebra, sometimes even lower in the pelvis, but more commonly at the level of the aortic bifurcation.

In order, therefore, to inidcate more graphically the variations of position that are most commonly observed, I have had a drawing made that will illustrate the most important types gathered from a review of the literature and from a study of the 25 personal cases that I am reporting (see Figs. 2 and 3). In the asymmetric type of renal fusion a marked degree of dystopia is the rule and the organ is commonly found lying at the promontory at the concavity of the sacrum or even far down in the pelvis behind the urinary bladder in the midline or at either side of the abdomen. This dystopia of the asymmetric horseshoe organ is so marked that many times it has been confused with a fibroid uterus in the female or a tumor of the sigmoid, easily palpable by rectal touch.

The weight and the size of the horseshoe kidney are variously described by the different investigators and may vary according to the associated pathology present; for example Legueu and Papin give about 300 gm., while Newman mentions 460 gm. and in our own 4 cases found at post mortem the weight has been from 280 to 350 gm., and hence a little more than the combined weight of two normal kidneys in the adult.

The organ as a rule is fixed and deeply incarcerated by its isthmus to the surrounding structures and blood vessels. In reality it is this lack of mobility that is responsible for its clinical syndrome. However, the two lateral halves and the superior poles that swing laterally to the lumbar region and which are sometimes widely open, may be freely movable, making surgical approach to either of the halves of this organ easily possible. The isthmus of the horseshoe kidney, together with the peculiar arrangement of the calices, renal pelves and ureters, constitutes the most characteristic feature of the renal fusion. The different types of isthmus have already been discussed. Anatomically the structure of the isthmus is of interest for its aberrant blood vessels, its gross size and its important relations, as we shall see later. Some authors have mentioned a third kidney formed from the enlarged and thickened isthmus, such as was observed in the cases reported by Rayer, Kuster and other investigators, and in one case of our own. But the phenomenon was in reality the retained fetal lobulation of the ventral

surface, so that the existence of the third kidney has never been demonstrated.

Histologically the isthmus is as a rule made up of solid renal parenchyma with cortical and some times medullary substance, microscopic sections of which reveal convoluted tubules and a number of normal or atrophic glomeruli.

According to Robinson, Beyer, Newman, Carlier and Gerard, Papin, Eisendrath and other writers, the isthmus joins the lower poles in about 90 to 95 per cent of cases and the upper poles in only about 5 to 10 per cent.

In our series there is only one case of this very rare variety, which was found at necropsy. It is illustrated in the drawing of Figure 5, where the fusion of the two kidneys occurs at the upper pole or more nearly at the upper middle portion of the two organs, forming an irregular letter H, with the isthmus lying across the vertebral column in front of the great vessels, and composed of solid renal parenchyma.

11. The excretory apparatus of the horseshoe kidney comprises the calices, the pelves and the ureters. These by their peculiar arrangement play the most important rôle as the typical anatomical structure, revealing the congenital malformation clinically and urographically. They are also the chief clinical and surgical factor in the causation of pathological conditions, ultimately giving rise to symptoms which reveal complicating infections and diseases due to lack of drainage.

Most writers and observers agree that the calices and pelves are as a rule markedly irregular in size, shape and position, owing mainly to the incomplete rotation of the organ as we have already seen in our embryological study. The pelves are generally two, situated ventrally or anterior to the blood vessels. They enter the horseshoe mass by the hilum independently of each other and hence constitute two separate organs. They are intrarenal or extrarenal and the variations in form, size and position are so great and so bizarre that one may say there are not two pelves alike even in the same case. The multiplicity of calices is the rule, and particularly striking is their reverse position looking inward toward the spinal column. The peculiar arrangement is most constant and characteristic in the lower calices, in view of their extension into the renal

isthmus and their consequent crossing in front of the vertebral column, as seen in the urographic studies. There are generally two ureters, one on each side of the vertebral column, passing across and in front of the renal isthmus to reach the pelvis in a peculiar angle. Because of their high implantation, it has been clearly recognized that there is a mechanical obstruction of some sort at the ureteropelvic junction, always resulting in the compression of the abdominal content by the overlying structure and in the production of retention, hydronephrosis, pyelonephrosis and other diseases in these kidneys.

Anomalies in the excretory apparatus of the horseshoe kidney are of quite common occurrence, the incidence of which is revealed by a brief survey of the literature. It is obvious that when the renal mass is ectopic in the bony pelvis, displacement and shortening of the ureters are a common observation. Cases of 1, 2, 3 and 4 ureters have been reported and observations of 1 to 4 pelves are also on record.

Anomalies in the location of the pelves are frequent. Kuster reported a case in which the right pelvis was found in the middle portion of the isthmus bridge with the ureter running almost perpendicularly between the aorta and vena cava.

Landouzy, Robinson and Durham have reported cases in which the ureters passed behind the isthmus. Carlier, Thacher and others noted a single ureter arising directly from the isthmus.

Losée reported the finding of a case with two left ureters which entered the bladder separately. Massari in 1897 reported a case with an ectopic ureter opening in the vulva. Robinson in the study of 55 specimens collected from the literature and from autopsy findings described a case of bilateral reduplication of ureters and pelves, showing 4 ureters and 4 pelves all entirely independent and opening normally into the bladder.

In our own series there is one case diagnosed cystoscopically and pyelographically in which 3 ureters opened into the bladder and 4 renal pelves could be seen in the pyelogram (see Figs. 26, 27 and 28). There is also a report of another case, which was found at post mortem, having 3 ureters opening into the normal bladder, as shown in the drawing of Figure 4. Later on, in the section on diagnosis, I shall discuss the rare occurrence of normally situated pelves in

the pyelographic studies of horseshoe kidney, and also the rotation of the calices and pelves without the presence of horseshoe kidney.

The presence of concomitant anomalies of other organs must also be emphasized, since many such cases are reported in the literature and appear to have some constant clinical bearing. Although the suprarenal glands are embryologically, functionally and anatomically in intimate relation with the kidneys, these organs according to various writers and post-mortem investigations always preserve their independent function and their normal anatomical position on either side of the spinal column even in cases of marked dystopia. Fusion of the two suprarenal glands may, however, occur, and cases of this type are on record. Thus Neusser has described a case in which a union of the two suprarenals occurred in the form of a horseshoe suprarenal. Kelly too describes a case of fusion of the two kidneys by the upper pole in an embryo with spina bifida, diaphragmatic hernia and fusion of the adrenal bodies. Many other anomalies of interest are frequently associated with fused kidneys, among which malformations of the genital organs appear to be more frequently observed than any other variation, owing mainly to the close connection between the müllerian and urinary ducts during the developmental stage.

Goddard for example reports a case of atresia of the rectum and patency of the urachus, and Ballowitz in 103 cases of renal malformations found 73 concomitant abnormalities of the genital organs. Quite common too are anomalies in the blood vessels supplying the horseshoe organs, as has been pointed out by Papin, Eisendrath and others; in fact aberrant vessels may be formed from any contiguous structure where the kidneys are lodged.

III. The blood, nerve and lymphatic supply of the horseshoe kidney merits a careful study on account of the anatomical, surgical and clinical significance that it presents, as follows: (*a*) The blood vessels, in addition to the physiological function of the circulation, contribute greatly to the maintenance of the position of the horseshoe organ, and therefore play an important rôle in the risk and difficulties to be encountered in any operative approach. (*b*) The nerve supply is responsible for the nervous irritability in the early symptoms and clinical manifestations that have been observed. (*c*) The lymphatics have perhaps been the principal etiological

route of infection and early pathological lesions of these anomalous renal masses.

(*a*) The blood vessels vary in position, size and number and are as a rule anomalous because, owing to their unique position, there are no two organs alike with the same arterial or venous supply and also because of the presence of anastomoses and collateral branches, as well as of superficial plexuses which have received extensive attention from many authors.

The blood vessels in most instances connect directly with the aorta and vena cava. As a rule there are from 4 to 6 renal arteries, extending laterally and caudally, two to each hilum to irrigate the corresponding half of the organ, and one or two to the middle portion of the isthmus (see Fig. 1). The two hilar arteries running from the aorta outwardly to the renal mass are sometimes subdivided into two or three branches of the pyelic and retropyelic arteries. The isthmic arteries also may be subdivided into two small branches, superior and inferior, coming sometimes directly from the aorta, sometimes from the common iliac, and sometimes appearing as collaterals from the mesenteric artery. However, in horseshoe kidney the anomalies in blood vessels are so numerous that they may be regarded as the rule.

Papin, studying from the literature 223 autopsy cases of horseshoe kidney, found and tabulated the following renal arteries: 1 artery in 1 case; 2 arteries in 26 cases; 3 arteries in 40 cases; 4 arteries in 25 cases; 5 arteries in 20 cases; 6 arteries in 9 cases; 7 arteries in 3 cases; 8 arteries in 2 cases; 10 arteries in 2 cases; and in 10 cases the authors stated a multiplicity of arteries, without mentioning the exact number. Papin finally stated that, in 189 cases studied, 86 had from 3 to 5 arteries, which is approximately two-thirds of the total cases.

In some cases, when the renal mass is low in the pelvis, some of the arterial branches run from the contiguous trunk, as the iliacs, the middle sacral and the hypogastric, and in rare cases anastomosis may be found from the spermatics, the mesenterics and the colics.

The important and close relation of the renal isthmus to the great abdominal vessels has already been mentioned. As a rule the bridge of the renal fusion is situated anteriorly, so that the posterior surface of the isthmus is marked with a furrow for the passage of the great

FIG. 7. Plain roentgenogram of Case IV, representing two enormous shadows of a coraliform calculus occupying practically the entire right and left pelves of a horseshoe kidney. Notice on the right the shadow of the calculi placed obliquely and overlapping the body of the third and fourth lumbar vertebrae. On the left side the lower calices are filled with the calcified deposits of the stone which also point inward to the midline, so that the plain roentgenogram makes the diagnosis of horseshoe kidney complicating the presence of bilateral nephrolithiasis. (Hernandez's Case.)

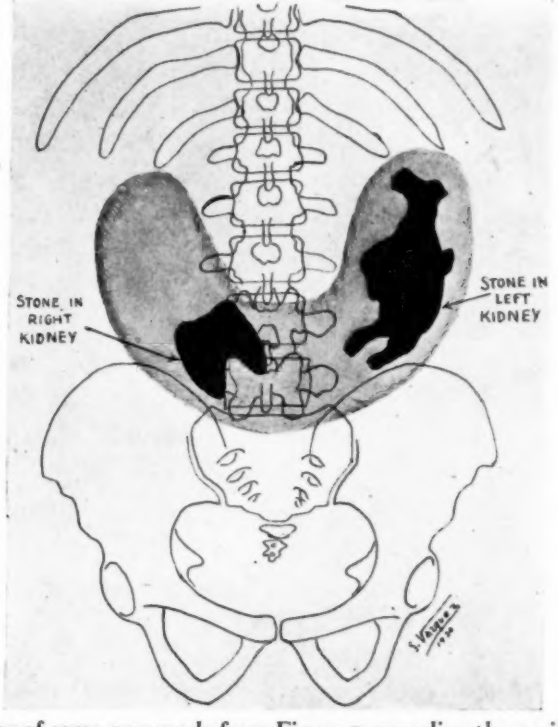

FIG. 8. Drawing of same case made from Figure 7, revealing the position of the horseshoe kidney in close relation to the vertebral column and bony pelvis, with the two enormous coraliform stones that were later confirmed by pyelographic examination.

FIG. 9. Bilateral pyeloureterogram of Case 1, revealing the inward rotation of right and left pelves and their close proximity to the vertebral column; also showing the peculiar position of the lower calices extending into the isthmus and revealing the presence of horseshoe kidney. This patient has been suffering from chronic constipation from birth and was acutely ill when he came into hospital with various erroneous diagnoses. Complete urological examination disclosed the presence of horseshoe kidney disease, which was treated successfully by the method of the indwelling ureteral catheter as shown in Figure 41.

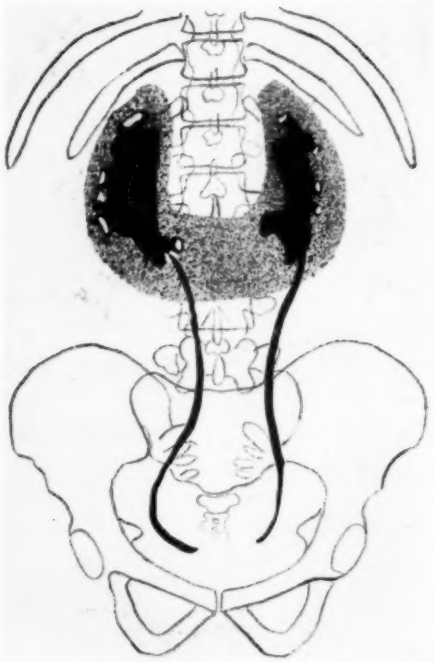

FIG. 10 is a drawing of Case 1, revealing the position of the horseshoe kidney and the peculiar arrangement of the inward rotated pelves and of the lower calices extending across the renal isthmus, which made possible a definite diagnosis of horseshoe kidney disease. Note that the ureters appear to come from behind. Clinically the horseshoe kidney syndrome has been markedly present for more than fifteen years.

vessels. However, instances have been observed in which the aorta and vena cava are anteriorly located, crossing the isthmus ventrally.

Nixon has reported a case in which the isthmus of the renal fusion passed behind the great vessels and Kelly described a case of fusion of the upper poles with fusion of the adrenals, all passing behind the great vessels and between them and the vertebral column. Carlier has emphasized the fact that in fusion of the superior poles this rare deviation from the posterior position of the great vessels is very conspicuous. Robinson in reporting a case stated that it occurs in 10 per cent of the cases, but it is obvious that this percentage is rather high when only 3 or 4 cases of this type are mentioned or described in the survey of the literature.

(*b*) The nerve supply of the horseshoe kidney is anatomically, physiologically and clinically of great significance, and only by studying its intricate topographical relationship can we come to explain the peculiarities of its clinical syndrome, which is not yet well understood.

We know since Cruveilhier gave its description that the nerve supply to the normal kidneys originates from the solar plexus and the branches of the splanchnic nerve; also that a network of anastomoses of the sympathetic and parasympathetic nerves is formed in the constitution of the renoaortic ganglions and renal plexus, which by terminal prolongation following the course of the renal artery reaches the hilum and penetrates into the small capillaries of the organ. But it is the retroperitoneal space, between the peritoneum and the vertebral column where the horseshoe kidney is lodged, that offers the most striking picture of the topographic and close relationships of the abundant nerve roots, too striking indeed to be overlooked in the causation of epigastric pain and the horseshoe kidney syndrome in its early clinical manifestation.

In fact the solar plexus originates from branches of the right and left semilunar ganglions, which are formed by branches of the splanchnic and phrenic nerves, and also by numerous plexiform branches directed transversely from one semilunar ganglion to the other, and making further anastomoses with the right pneumogastric or vagus nerve. Still further interanastomoses and plexiform branches are formed in front of the aorta, giving exit to the renoaortic ganglions and celiac plexus, the intermesenteric plexus,

FIG. 11. This drawing represents the anatomic structure and topographic relation of the organs situated in front of and behind the isthmus of the horseshoe kidney, and serves to point out the important relation to the blood, nerve and lymphatic supply and the etiological rôle played by these in horseshoe kidney disease. Notice that, in a section made in a horseshoe organ where a great portion of the isthmus could be removed, the organs situated behind it are bound together and incarcerated by the pressure and tension which the isthmus of the horseshoe organ constantly exerts by its weight, its lack of mobility and the intra-abdominal pressure it exerts, which are responsible for the characteristic horseshoe kidney syndrome that has been observed clinically in practically every case of this series. (Modified drawing from Testut and Jacob's Anatomy and Papin's Chirurgie du Rein.)

A. Phrenic nerve.
B. Splanchnic nerve.
C. Semilunar ganglions.
D. Reno-aortic ganglions.
E. Pecquet's cisterna.
F. Sympathetic nerve.
G. Vena cava.
H. Aortic lumbar plexus.
I. Pneumogastric nerve.
K. Splanchnic nerve.
L. Semilunar ganglions.
M. Reno-aortic ganglions.
N. Renal plexus.
O. Thoracic duct.
P. Sympathetic nerve
Q. Aortic artery.

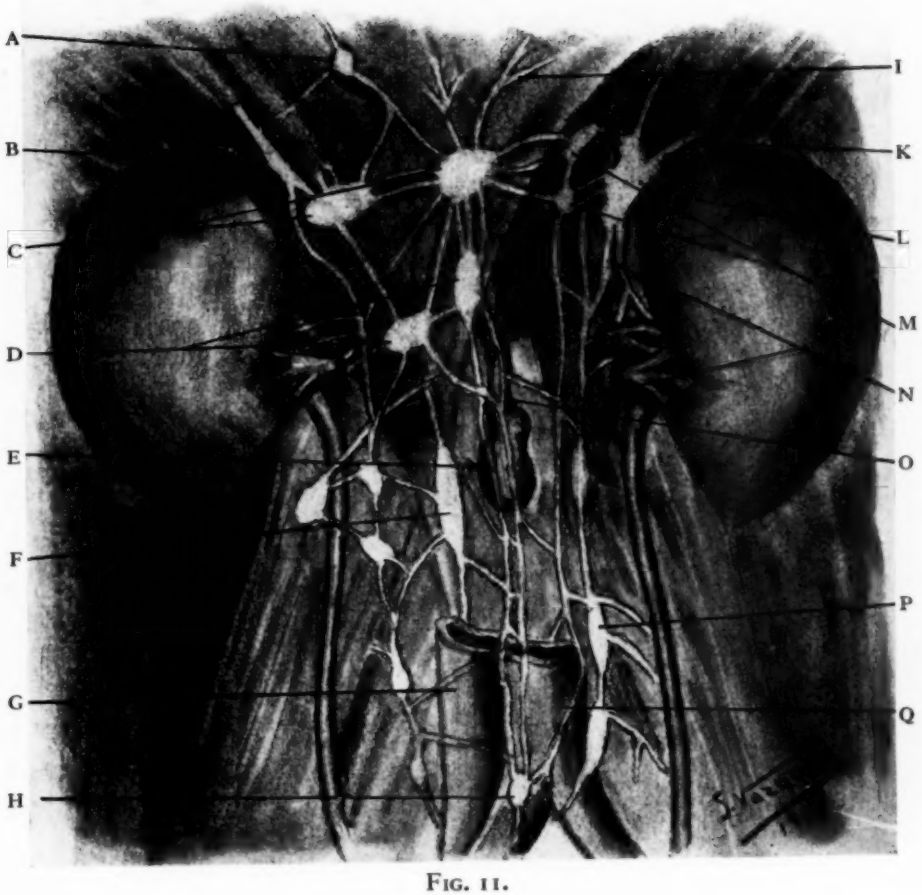

FIG. 11.

and finally the richly organized and sensitive solar plexus, which is of paramount physiopathological importance not only because it sends branches to almost all the viscera of the abdomen but also because of its exquisite sensitiveness and response to sensations, pressure and irritations, expressed in sudden pain and gastro-intestinal disorders, producing so-called epigastric or umbilical pain. Hence it is logical to assert that beyond a doubt the continuous pressure exerted by the weight of the isthmus in cases of horseshoe kidney upon these anatomo-physiologically important structures of blood vessels, nerves and lymphatics, will account to a great extent for the persistency of the chronic suffering and the clinical syndrome of this potential clinical entity. The relations of these structures are brought out graphically in Fig. 11, a modified drawing after Testut and Jacob, Papin and others.

(*c*) The lymphatic circulation is also of considerable importance in the pathogenesis and clinical development of horseshoe kidney disease. First of all we have the multiplicity of lymphatic ganglions which essentially constitute the lymphatic system of vessels, situated in the cellular retroperitoneal space, at the renal pedicle and running parallel on both sides to the great abdominal vessels, where they lie in the form of a chain, connecting the lymphatic circulation of the iliac group of ganglions with that of the lumbo-aortic. In addition to these we must not forget that behind the aorta and vena cava and between the vertebral column and the great vessels, lies the mother lymphatic reservoir of the chyle, Pecquet's cistern, which may also be cut off, and therefore affected physio-pathologically, by the extra pressure of the isthmus of the horseshoe renal mass in front of it, interfering with the normal circulation of the lymphatic ducts and the chain of lymph-nodes (see Fig. 11). Horseshoe kidney would thus play an important etiological rôle in the stagnation of lymph, producing infection, symptoms and diseases of the organs of the retroperitoneal space where the horse-shoe kidney holds a prominent place. An example of this patho-genetic condition is readily seen in the third-case of the 4 autopsy cases I am herewith reporting. (See Case XXIV)

Briefly, the case is that of a young man thirty-five years of age who was admitted to the medical wards of the New York Hospital complaining of abdominal pain with slight frequency of micturition and who, after a month of observation, was submitted

to an exploratory laparotomy for an undetermined abdominal tumor, which proved to be a retroperitoneal lymphosarcoma. Three weeks later at post mortem a horseshoe kidney was disclosed with an enormous lymposarcomatous mass lying at the thoracic duct or Pecquet's cistern, densely adherent and covered by the isthmus of the fused horseshoe renal mass, with many metastases and marked emaciation that proved to be the direct cause of his death.

IV. The topographic relations of the horseshoe kidney have not been well studied and are certainly of capital importance. The relations vary mainly according to the position in which the organ may be found and the type of horseshoe kidney encountered, and have great significance for the pathogenesis of early clinical manifestations and for the surgical management of this condition.

The most important anatomic structures and topographic relationships of the congenital anomalous organ have already been discussed in Sections I, II and III; so I shall at this time merely point out the rôle of the close relationship to the peritoneum and its abdominal contents.

Although, like normal kidneys that are normally placed in the lumbar region, the horseshoe kidney is well protected by the enveloping prerenal and retrorenal fascia of Zuckerhandl, and although it is still well surrounded by the dense fatty capsule, which preserves it from injuries and facilitates the physiological movements during inspiration and expiration, the isthmus in this anomalous kidney lying in front of the great vessels in the cellular retroperitoneal space, lacks the presence of the renal fatty capsule, so that its mobility is reduced to a minimum and the kidney becomes virtually a true and permanent fixed organ resting in the middle of the abdomen, incarcerated by the surrounding structures and pressing directly upon the circulation of the great vessels, upon the solar plexus and its many vasovisceral branches and also upon the lymphatic circulation of all the organs of the abdominal cavity and of the lower extremities.

The parietal peritoneum with its posterior layer exquisitely supplied with lymphatics and nerves lies directly upon the fused bridge of renal parenchyma and is only separated by a thin layer of the prerenal fascia, which crosses the midline to continue with that of the opposite side, but without the perirenal fatty capsule. The peritoneum is therefore densely adherent to the anterior surface of the renal mass as well as to its excretory apparatus, the pelves

and the ureters, which are also ventrally situated. Undoubtedly under these conditions the intra-abdominal pressure and the weight and close relation of the intra-abdominal organs which are bound together upon it—such as the duodenum, the head of the pancreas, part of the stomach and liver and sometimes the transverse colon, or even, when the organ is low in the pelvis, the sigmoid, small intestine, epiploon and other intra-abdominal organs closely related to it—must play an evident rôle in the urinary stagnation and early clinical manifestations, as has been definitely observed in this series of cases.

The reflex response of the solar plexus to the continuous insult would then produce intestinal motility and increase of peristaltic waves with irregularities in the dynamic contraction of the ureters and pelves, bringing on a marked stagnation characterized clinically by a history of intestinal disorders with chronic constipation and intermittent attacks of pyelitis and pyelonephritis, as we shall see later in the description of many of the cases here reported.

VI. Etiological Factors

In considering the etiological factors of horseshoe kidney disease, the type of renal symphysis that we shall discuss is the one most commonly observed clinically, namely the symmetric type of renal fusion by the lower poles with the concavity facing upward and the isthmus lying across the midline of the abdomen in front of the great vessels. In this disease the incidence of age and sex has not played any important part, although in our series of 25 cases, herewith reported, 17 of the patients were males over eighteen years of age and 8 were females ranging in age from twenty to forty years, with an average of thirty years and a greater percentage of the male.

The horseshoe kidney may be found at any age, but as a rule it is not discovered clinically until a superimposed infection sets in, since the signs and symptoms are overlooked in childhood and the diagnosis in this condition is seldom made. The principal factor in the disease, even in normal horseshoe kidney, is the chronic irritability of the organ itself as well as of the structures in its vicinity, particularly the parietal layer of the peritoneum because of its vascular supply. The continuous irritation of this serosa and other neighboring tissues and structures during the normal

movements of the body tends to produce a certain degree of potential irritability which naturally results in tissue inflammation and stagnation of both the urinary and the gastrointestinal tract.

The unique position of the isthmus lying across the midline is mainly responsible for this irritability because of the pressure which its weight exerts upon the abdominal organs, both those retroperitoneally and those intraperitoneally situated. These organs are so bound together and are so closely adherent that the increased abdominal pressure to which they are subjected is sure sooner or later to cause rectal and vesical symptoms which, combined with the characteristic epigastric or umbilical pain, constitute the true horseshoe kidney syndrome.

Furthermore as a result of the abnormal anatomic position of the pelves and ureters, which are situated ventrally and pass across the isthmus of the anomalous kidney, the rhythmic dynamic contractions and physiological movements of these organs are sooner or later interfered with, causing the development of pyelitis and peripyelitis, of ureteritis and periureteritis. These conditions gradually extend to both ends of the ureters, to become, after a chronic process of long standing as in chronic nephritis, an acute or exacerbated one, characterized by pyelonephritis with some urinary stasis, back pressure and consequent cystitis, for which the patient is finally examined. A pyelographic study will then reveal the presence of horseshoe kidney disease in its essential purity, before the presence of a gross concomitant pathology can be established.

As the early symptoms progress the clinical picture may become aggravated, with the general system also becoming involved, essentially through lack of drainage, and through intense pyelonephritis, followed by recurrent attacks of fever, chills, general sepsis, ileus and uremia. In our series, half of the patients have been more than once critically ill, and one of them, a medical student, died with the clinical description given here. These facts, like those in polycystic kidney disease, reveal that the occurrence of renal failure is bound to be more common than is generally expected.

At the same time other clinical manifestations may arise as the result of this condition. The isthmus of the horseshoe organ is deeply fixed and practically immobile, resting in its prominent position across the vertebral column, and the direct pressure it exerts

upon the great abdominal vessels, more pronounced for lack of a fatty capsule at the renal bridge, offers anatomically conclusive evidence of the etiological rôle played by the faulty circulation of the extremities and abdominal viscera, which may be a concomitant factor in the development of the associated pathology. The lymphatic and the nerve supply may also be interfered with as a result of the traumatic body insult, the lymphatic interruption resulting in stagnation of the lymph in its mother reservoir, the cistern of Pecquet, while the nerve interference causes over-stimulation of the most exquisitely sensitive area of the solar plexus and its visceral branches. Furthermore, as the isthmus rests upon the aorta, the continuous rhythmic contraction of this great artery in systole and diastole affects the organ and tends to produce symptoms of aortitis and even aneurysm, as has been reported in several instances in the literature and as was observed in one of the 4 autopsy cases in this clinical study. Accordingly it is common to observe compensating cardiac hypertrophy, thrombosis of the iliac veins and phlebitis of both legs for lack of circulation as the result of the horseshoe kidney disease.

All these etiological factors and their vivid clinical pathology convince me of the fact that we are dealing here with a true clinical and anatomo-pathological entity which must be recognized as the horseshoe kidney disease.

(*To be continued*)

The American Journal of Surgery

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EDITORIALS

LEST WE FORGET

FOR the past two decades two men have been prominent in American obstetrics. They were the generals leading the forces of conservatism in midwifery. In their writings, daily contact with students and practice, both John Osborn Polak and J. Whitridge Williams were the generals of the forces of sanity in this branch of medicine.

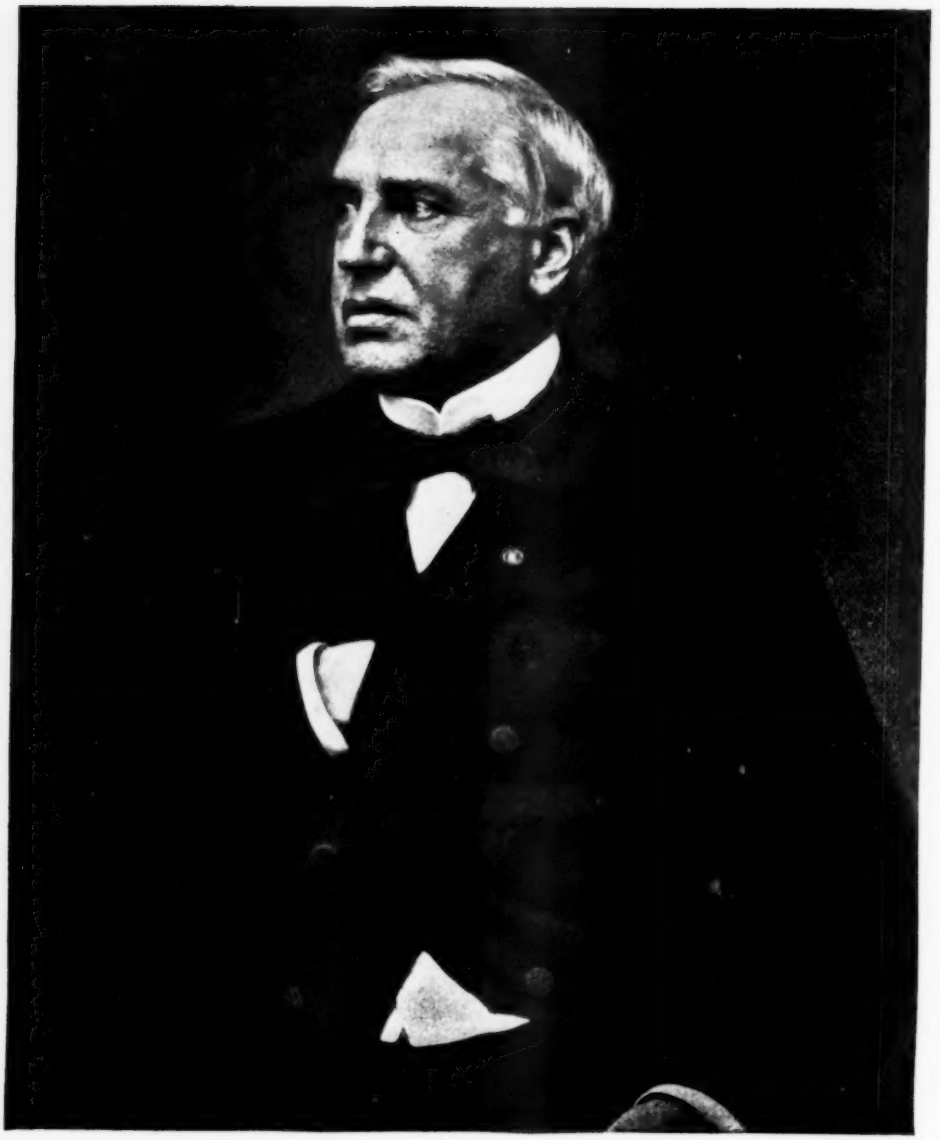
While in this country the published mortality records are nothing to point to with pride, these gentlemen, in their efforts to make childbearing less dangerous, continued to preach and drive home the fundamental lessons of giving nature a chance, eschewing the bizarre and radical. They believed that more hours should be devoted to the teaching of obstetrics in the undergraduate schools. They affirmed that students should have an opportunity for a wide acquaintance with the clinical side of obstetrics in the delivery-room and prenatal clinic. They were insistent that all teaching should emphasize time-tried, conservative measures. When obstetricians of more or less reputation advocated short cuts to delivery, for the most part entailing meddlesome midwifery, Polak and Williams claimed the methods were to be condemned because it gave the neophyte a set of false values which would result in human havoc later in their professional careers.

It has been common knowledge that there are some in this country who have

sought publicity by suggesting all sorts of fantastic procedures, procedures aimed to shorten labor and effect rapid birth. But these men have never come into the open and dared the fight. Whenever they lifted their heads above the scientific hedge Polak and Williams were not slow to hit and in scathing terms condemn and so prevent the questionable ideas from gaining a foothold and becoming popular with the common run of practitioners, who get their ideas from the higher-ups.

We sit back and wonder if these men of radical tendencies will now bravely run into the open and deluge the profession with a thousand and one new ways of terminating normal labor. It would not surprise some if in their zest to get the spotlight will go to the great length of advocating cesarean section in all cases. Those who believe in conservative obstetrics must take up the cudgels so ably handled by Polak and Williams and continue the fight for sane methods.

T. S. W.



AUGUSTE NÉLATON

[1807-1873]

The American Journal of Surgery
N. S. Vol. XIV, December, 1931

NÉLATON'S LINE

Auguste Nélaton was born in 1807 in Paris. He presided with Malgaigne, who has been described as the greatest surgical historian and critic the world has yet seen, at the Hôpital St. Louis. He comes down in history as an operator and teacher with a reputation as great as that of Dupuytren. We are told that he was a most modest man, quiet in his habits, of a friendly disposition and most considerate of the unfortunate.

He was the first to devise a bullet-probe (this was used in Garibaldi's case). The present-day flexible rubber catheter was his invention. In gynecology, he was the first to describe pelvic hematocele (1851–52), and due to his efforts, ovariotomy was established in France. His principal work is his "Éléments de Pathologie Chirurgicale" (1844–59). The surgeon is familiar with the Nélaton dislocation, the wedging of the astragalus between the widely separated tibia and fibula, usually complicated with fracture. The fibrous tumor or sarcoma lying between the peritoneum and the muscles of the abdominal wall is known as Nélaton's tumor. Nélaton's line is a line drawn from the anterior superior spine of the ilium to the tuberosity of the ischium; normally the great trochanter lies in this line, but in cases of iliac dislocation of the hip or fracture of the neck of the femur, the trochanter is felt above the line.

Auguste Nélaton died in 1873.

T. S. W.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

A CHAT WITH AESCULAPIUS

W. B. Howell, M.D.

MONTREAL, CANADA

OF THE thousands of medical men who visited the Carver clinic at Malad City, Utah, during the lifetime of the founder, Dr. Jimmy Carver, there can have been few who did not come away with the impression that the great surgeon was absorbed in his professional work to the exclusion of all other subjects. He had, nevertheless, a hobby in which he was profoundly interested. This was the study of the language, history and literature of ancient Greece. Great though the demands upon his time were during the last twenty years of his life, he made it a rule to devote at least an hour every day to this diversion. His annual holiday, which he took in the spring and lasted three months, was almost invariably spent in a trip to Greece. There were not many parts of that country that he had not visited at some time, on foot, on horseback, or in a motor car.

During one of these trips he found his way to a small village on the shore of the Gulf of Salonika, and started off alone to walk up the slope of Mount Olympus. It was a fine spring morning. The sun, however, was already giving promise of the coming heat of the Greek summer. The path wound steadily upwards through woods and shrubs and across open spaces where the ground was carpeted with brilliantly colored flowers. Now and then he stopped to look down upon the "wine dark sea," or to feast his eyes upon the majestic beauty of snow-covered Olympus. There were ecstatic moments when the beauty of the scenery made him feel that he was indeed approaching the home of the gods.

After walking for two or three hours, and meeting no one, he came suddenly upon a small temple, partly in ruins and shaded by a few plane trees. There was, over the doorway, an inscription almost effaced by the hand of time. Carver stood for a while trying to read it, but the only word he could decipher was "Aesculapius." He entered the little building and finding its shade very agreeable after the heat and glare of the midday sun, sat down upon a stone bench which was built along the wall on one side of the entrance. Tired with walking he leaned his head against a stone pillar and momentarily closed his eyes. When he opened them he found to his astonishment that he was not alone. Sitting opposite on a bench similar to the one upon which he himself was seated, was an old man with a shining bald head, and

long beard. He was clothed in a garment of snowy whiteness, draped about him in a manner which reminded Carver of an ancient Greek statute. There was a certain grace about its flowing robes in spite of the fact that the body underneath was obviously short as well as corpulent. Beside him, propped against the bench on which he was sitting, was a curiously carved staff. In halting modern Greek, Carver said that he hoped his presence was not an intrusion. His astonishment was great when the old man answered in English, "Sir, I could never look upon the presence of a member of your profession as an intrusion."

"Well, that's very kind of you," said Carver, "but how do you know what my profession is?"

"It would be a strange thing," answered the little man, "if I could not recognize a doctor when I saw him."

"Oh," said Carver, somewhat puzzled, "you are probably one yourself."

"I was—alas I am not now."

"You have retired, I suppose. Well, we all come to it if we live long enough. I shall be giving up myself in a few years. Where did you practise, doctor?"

"I practised my art in many cities and in many countries; in Greece, Asia and Egypt—even in India."

"Well," said Carver, "I've stayed put in one place. Its kind of hard to make a new start."

"I moved from place to place, not because I wanted to, but because I was forced to. My fellow physicians were always jealous of me, and spread reports that I belonged to the hated class of goetes.[1] They roused the mob to set on me and kill me, and often I had to flee for my life. It was my power to raise the dead to life that made my rivals angry."

[1] A class of wizards in ancient Greece who were feared and hated. "They were the confectioners of philtres and poisons; they peddled their craft for money; they raised jars, jealousies, strifes, like a thick scurf o'er life." (Montague Summers: Geography of Witchcraft. Lond., Kegan Paul, 1927.)

"Poor fellow," thought Carver, "I wonder if he has general paralysis or paranoia." Then he said aloud, thinking to humor his new acquaintance, "Yes, they wouldn't like you raising the dead—especially those who had been their patients. There might be a demand for explanations. But tell me, how did you restore the dead to life?"

"Ah, many have asked me that question. It is one of the secrets which I have no right to impart. Some of us knew how to do things which you cannot do now, for all your boasted progress. But in the end my secret was my undoing. It has always been believed that Pluto thought that I was defrauding him of his rights, and instigated Jove to strike me dead with a thunder-bolt. It wasn't that at all. It was the heirs of the people I brought to life who thought that I was defrauding them. And they didn't go to Jove about it either. They acted for themselves; that's how I came up here. Besides, the dead themselves were none too pleased about being restored to life. They complained that it was no joke to have to die a second time."

"Say, doctor," asked Carver, with a feeling of growing interest mixed with amusement at his companion's fancies, "would you mind telling me who you are?"

"I am Aesculapius," the old man answered simply.

"Oh yes, Aesculapius; yes, of course, I should have known that," replied Carver tactfully.

"You do not believe me, but my name is Aesculapius as certainly as yours is Carver."

"Tell me how you know my name."

"I have seen you working at Malad City. You see it is one of my pleasures, indeed one of my greatest pleasures, to go about among mortal men from time to time to see how the art of medicine is progressing. It was an art when I practised it. Not so very long ago I thought it was going to be a science, but now I see that it has become a business." He sighed, and then went on, "You have built up a great

clinic. You minister to thousands of sick people—you should be a happy man."

"Yes, I have made a success of the clinic. But happy—well, I don't know. I suppose I am as happy as most people. But I don't seem to remember your face, doctor."

"That is not to be wondered at," was the reply, "for when I go about among men I do not appear as you see me now. I seek out some physician or surgeon who is on the point of setting forth upon a round of visits to medical centres or to some convention, and I borrow his body and his name. I lull his spirit to sleep and hide it in the heights of Cytherea or Idalium.[1] I go in his likeness and no one knows."

"Doesn't he raise any objections?"

"When I return his spirit to his body I scratch his right ear[2] and he remembers nothing—not even that he didn't go. He never admits that he does not remember what happened during his trip. If he did admit it he knows what people would think, and his wife would never let him go away again, not alone at all events."

"You must have a great love of medical work, doctor," said Carver.

"I have," answered Aesculapius. "I am not only the god of medicine but the inventor. It is to me as a first-born child. But it is not only that—you must realize that we immortal gods find the time hangs heavily on our hands. We have so much of it. We can't always be playing golf or bridge; it takes a fool to do that. Everything we do we remember having done before, not once or twice, but thousands of times. The fact is, immortality is spoiled by memory, and yet without memory we would have no immortality. You mortals don't realize that your greatest blessing is death, and are silly enough to be afraid of it." A look of weariness came into the face of the little old man. He yawned and continued, "It is the infinite variety in disease which makes me long to be a physician again."

"Isn't there any opening for a hospital among the gods?" asked Carver.

"Alas no!" said Aesculapius, "I often wish there were. Not that there is no disease among them, but it never gets worse—and nothing can make it any better. Bacchus, now, has cirrhosis of the liver. You know what his habits have been; he has ascites, but it never gets any better or any worse. Once when I first came up here I tapped him. How green I must have been! You wouldn't believe the fuss the Fates made! They're the only people among the immortals that grandpapa's[1] afraid of. They told him. Well, the old gentleman came down here in a desperate hurry to see me about it. I don't like to think of that time. And then there's Silenus, the old dipsomaniac. He can't leave the nectar alone, and never could. I should like to try the effect of pilocarpine on him. No wonder Bacchus got the habit with such a tutor. I used, too, to want to do something for Polyphemus's eye. Ulysses made a dreadful mess of it. And it wasn't only his eye; his digestion was permanently impaired. He gorged himself on Ulysses' friends and drank too much of that miserable Greek wine.[2] I don't know whether it was the Greeks or the wine, but he hasn't been able to keep a Greek on his stomach since. Polyphemus hasn't had much of a life since those days—blind and dyspeptic. And then there's Vulcan with his malunited fracture. It would not be easy to reset; the bones of the gods are very hard. Yes, I've often thought we could have an excellent hospital. We'd have a nursing school and train nymphs and naiads. They would be much better

[1] "But to prevent suspicion, will I steep
His temples in the dews of balmy sleep,
Then to Cytherea's sacred seats remove,
Or softly lay him in the Idalian grove."
(Virgil: Aeneid. Bk. i, 11, 680–681; Christopher Pitt's translation.)

[2] "The seat of the memory lies in the lower part of the ear." (Pliny: Natural History. Bk. xi, chap. 106.)

[1] Aesculapius was the son of Apollo, who was the son of Jupiter.

[2] "Ejecting from his monstrous maw
Wine mixed with gore, and gobbets raw."
(Virgil: Aeneid. Bk. iii, 11, 632–3. Conington's translation.)

off employed in a hospital than roaming about the woods and streams without a chaperone and getting into trouble with the fauns. We'd do as you do in America; let them think they're college students. We'd give them plenty of lectures and encourage them to wear class pins and blazers and have a college yell. And every little while we'd give them a degree. I'd let them have a dance now and then and ask their faun friends. It is too bad that Argus isn't living. We could have employed him to watch the nurses at the dances—fancy a chaperone with a hundred eyes—and what a hall porter he'd make for night duty!"[1]

"You wouldn't need Argus," said Carver, "if you put the training of your nurses into the hands of the Graces. Their influence, I am sure, would—"

"Yes," interrupted Aesculapius, "their influence would be excellent, but the trouble is that they never wear any clothes. I am afraid it wouldn't do at all—such a bad example for the nursing school and so disturbing for the patients. We could get Neptune," he continued, "to take charge of the department of hydrotherapeutics. His sea nymphs could be trained to help him. Aunt Lucina[1] could take charge of the maternity wing, and Ceres would, of course be our dietitian. And think of the advantages of having centaurs trained in first aid for the ambulances!"

"How about Hercules?" asked Carver, "A strong man is invaluable about a hospital sometimes. Didn't Eurysthenes find him very satisfactory as a chore man?"

"Oh, we could find him better employment than doing chores," replied Aesculapius, "I'd give him charge of the department of remedial gymnastics. He'd be glad of something to do, and then he'd be useful in keeping the house doctors in order. He would like the exercise. He's getting disgracefully fat."

"Your staff would be a highly efficient one, doctor," said Carver, "but it seems to me as if it would be rather out of proportion to the number of patients you seem likely to have. After all, a hospital exists for the patients."

"Not necessarily; you know that even in America patients aren't always the first consideration. Some hospitals exist for the benefit of a university, some for the advancement of science, some for the good of the medical staff, and some to impress visitors. I know hospitals which exist, as far as I can see, for the benefit of the nurses, and one or two apparently, for the orderlies. I daresay," he continued, "grandpapa wouldn't mind very much our having a hospital if we asked him to be president. Of course, we'd have to watch him when he made his visits. We should have to keep the nurses out of sight. There has been at one time or another a good deal of scandal about him. You remember the episode of Io? That sort of thing wouldn't do in a properly conducted hospital—fancy the old gentleman petting one of our nurses and then turning her into a cow when he saw Juno coming!"

"You haven't said anything yet, doctor," said Carver, "about your department of surgery."

"No, of course that would be a great problem. I don't know how we could ever get a surgeon into Heaven. As we wouldn't have any deaths," Aesculapius went on, after a pause, "we wouldn't need much in the way of a pathological department. We could get Pluto to give us some ground in the infernal regions to build a department of research. I used not to like him at one time, but we have made up our quarrel. His place isn't far from here. He'd let us use Cerberus for experimental purposes if we put it up to him tactfully. Think of the advantages of working on a dog with three heads."

[1] This Argus had an hundredth eyes:
of which by turne did sleepe
Always a couple, and the rest did
duely watch and warde
And of the charge they took in hande
had ever good regarde
Ovid: Metamorphoses. Golding's Translation.

[1] Lucina was a daughter of Jupiter, and presided over the birth of children.

"Wouldn't he be rather large and fierce to handle?" asked Carver.

"Yes, perhaps. But you could give him a preoperative sedative,[1] and then Morpheus and Somnus could put him to sleep. They'd make excellent anesthetists. It wouldn't do any harm to amputate the third head and it would be easier for the anesthetists. It would cut down his barking by a third. The antivivisectionists would make a fuss, but—"

"Do you mean to tell me," interrupted Carver, "that there are antivivisectionists in Hell?"

"My dear sir, it is crowded with them, and they are just about as reasonable there as they were on earth. If you were to try to do anything for the benefit of science to one of the vultures which have been tearing Tityus' liver for these thousands of years the antivivisectionists would make your life in hell a burden. They don't mind Tityus' feelings, though."

"Tell me, doctor," asked Carver, "what sort of a place is Hell? Is it so very terrible?"

"No, not particularly so," was the reply, "there are a great many delightful and amusing people there. It is a little dark in spots and at times quite sultry. Tartarus is the worst part of it.[2] That is where the abortionists go, and those who have performed unnecessary operations for the sake of the fee."

"Those who split fees, too, I am sure," interposed Carver, who was a Fellow of the American College of Surgeons.

"No," answered Aesculapius, "not unless they have done worse things than that. Pluto had to build an addition to Tartarus lately; it was getting so dreadfully overcrowded. Some of the most distinguished of your former colleagues are there. But perhaps you would like to see the infernal regions. I have the entrée and will be glad to take you there for a visit. Shall we go now? The entrance is quite near."

He stood up. For the first time Carver noticed that the staff which the little man held in his hand had a living snake wreathed spirally around it.

"Well now, doctor," said Carver, with a shade of nervousness in his voice, "I don't know about going now." He looked at his watch and continued, "Maybe I should do better to leave this visit for another time. My friends will be expecting me back. I want to tell you," he went on hurriedly, as if afraid of being interrupted, "I have enjoyed making your acquaintance and having this visit with you. If you ever come to the United States again, and are near Malad City I'd like you to —" He stopped. He had become suddenly aware that he was alone. At what precise instant the little figure had disappeared he could not say. Carver rubbed his eyes. The afternoon was far advanced. The sun was sinking behind the snowy top of Olympus. He knew he would have to start at once if he were to reach his camp before nightfall. He stooped to pick up his walking stick which had fallen upon the ground. As he did so a snake which was lying beside it, and partly over it, glided swiftly away.

[1] "Arrived, they first grim Cerberus survey
Stretched in his den th'normous monster lay.
His three wide mouths, with many a dreadful yell
And long, loud bellows shook the realms of hell.
Now, o'er his neck the starting serpents rose
When to the fiend the dame a morsel throws.
Honey and drugs, and poppy juices steep
The temper'd mass with all the powers of sleep.
With three huge gaping mouths, impatient flies
The growling savage, and devours the prize.
Then by the charm subdued, he sunk away
And stretched o'er all the cave, the slumbering monster lay."

(Virgil: Aeneid. Bk. vi, 11, 420 et seq. Christopher Pitt's translation.)

[2] "One of the regions of hell, where according to the ancients, the most impious and guilty among mankind were punished." (Lampriere's Classical Dictionary.)

BOOK REVIEWS

THE RENAL LESION IN BRIGHT'S DISEASE. By Thomas Addis and Jean Oliver. N. Y., Paul B. Hoeber, Inc., 1931.

To say that the classification of Bright's disease has been found confusing and unsatisfactory to students, clinicians and pathologists does not begin to tell the story of the constant bane it has been to them. And in the attempt to arrive at some significant correlation between the findings of all manner of clinical investigations on the one hand and the nature and extent of its renal lesion on the other, the despair has only increased with the efforts put forth. Starting in the time of Bright, this now century old state of the matter has been frankly acknowledged and decried recently by such men as Christian and Schlayer, as it was earlier by the minds of such workers as Volhard and Fahr and still much earlier by such as Gull and Sutton.

Now come two men, a pathologist and a clinician, each of the highest repute and achievement in its research fields of his respective subject, who have pooled their lifetime interests and endeavors in this problem and have used an approach and an attack which, if not altogether different from those of the past, are at least fresher, certainly much more penetrating and give every promise of being infinitely more fruitful.

They have removed so well the many vitiating variables from clinical observations that their methods bear the cold white light of mathematical precision. Indeed, they have subjected these clinical items to an analytical technique of superb quantitative exactness, and with such methods and an unerring insight that must compel every scientific respect, they have come to a classification that is simpler, more understandable and of much greater clinical and pathological significance than these with which we have labored and wandered in the past.

More than that, they have submitted interpretations of durable acceptability; correlations and explanations which may well lead to firmer ground than we had yet hoped to attain in our knowledge of Bright's disease.

On the clinical side they have used principally the criteria of the ratio:

$$\frac{\text{Urea excretion (mg. per hr.)}}{\text{Blood urea concentration (mg. per 100 c. c.)}'}$$

and quantitative studies of the urinary sediment. They have used several ingenious conditions of control which reduced the sources of error and vitiation to a minimum. On the pathological side the study was limited quite strictly to morphology.

By observing the clinical phenomena in the manner they have outlined, they could "follow all the major changes in the extent of the renal lesion" and could "plot the whole history of the increase or decrease in the extent of the lesion from its inception to its termination."

Of particular interest to vital statisticians is the view that "probably even now the majority of all instances of Bright's disease remain unknown and unsuspected. It is only through . . . examining urine of apparently normal individuals that we are beginning to appreciate the wide prevalence of the disease and the great variety of its forms and to understand how relatively infrequent is that type of lesion . . . which carries with it any apparent consequence for the life of the individual."

A most valuable incidental product of this research is the system of quantitative standards which the authors have compiled for the arbitrary separation of persons in health from those with Bright's disease. By taking values which lie outside the range of normal variations (a protein excretion of more than 30 mg. per twelve hours, and a rate of cast excretion in excess of 5000 per twelve hours) they have drawn a dividing line of practical clinical value.

The result of their inquiry has eliminated the idea that the lesions in the hemorrhagic group are entirely inflammatory, in the degenerative group only degenerative or in the arteriosclerotic group solely arterial and atrophic. They seem to have established that glomerulitis is always present in the hemorrhagic group and never present in the degenerative or arteriosclerotic groups.

The plates are exquisitely done. In order to bring out certain details an unusually large page was necessary and this has resulted in a format that is characteristic of many of the monographs published by the same house.

A great deal more could be said of this book but it is difficult to say anything except in words of praise and the writer cannot help agreeing with the reviewer in the *Lancet* who felt that "to praise it would be an impertinence."

Urological Roentgenology. A Roentgen Atlas of The Genitourinary Tract with Case Histories and an Outline of Urology in Its Relations to Roentgenology. By Hugh H. Young, M.D., and Charles A. Waters, M.D., Assisted by Mary A. Goldthwaite. Illus. by William P. Didusch. Ed. 2 Revised. New York, Paul B. Hoeber, Inc., 1931.

This magnificent work of some 500 pages and some 600 illustrations forms Volume 7 of the well known series of monographic atlases in roentgenology edited by James T. Case. In all propriety it can be designated a monumental work, both because of its conspicuous supremacy as a text and atlas in urological roentgenology and because it stands as a veritable monument to the clinic from which it emanates.

The book serves essentially a twofold purpose. First, it brings to the reader practically everything in the way of a post-graduate course in urological roentgenology that is possible through the medium of words, photographs and sketches. Incidentally, much of the text extends to considerations quite outside the scope of roentgenology, though the close relationship is ever obvious. Again incidentally, its medium of photographs makes of the course provided by this book one peculiarly close to a personal course since roentgenology is, after all, primarily a study of photographs. Second, the book is a diagnostic reference guide, to which one might turn as readily and almost as profitably as to a personal consultation with the authors. This feature is especially facilitated by the diagnostic index to case histories. Here one can find numerous histories and a wealth of data on cases similar to any particular case of his own that may present certain diagnostic puzzles.

The division of subjects into chapters is as follows: technical considerations; normal landmarks, normal measurements and normal functional findings; renal anomalies; obstructive conditions; infections; tuberculosis, lithiasis; tumors; traumatisms and foreign bodies; neurological disorders; intravenous urography; arteriography; recent progress in technique.

The case histories are so concise, so limited to essentials, and so much use is made of well understood abbreviations that the histories have been very conveniently placed so as to fill about one-third of a page at the bottom of each illustration.

One of the finest features of the book is the superb art of Mr. William Didusch, who has contributed the sketches and colored plates. A view of Mr. Didusch's work makes quickly apparent the reason why he is so generally esteemed as having no peer in America in this field of art.

Diseases of the Gums and Oral Mucous Membrane. By Sir Kenneth Goadby, K.B.E., M.R.C.S., L.R.C.P., D.P.H. Oxford Univ. Press, 1931, 496 pp.

The author in his Preface points out that at the time of the publication of the first edition "focal infection as the origin of serious disease was regarded as academic, though interesting." And he adds the following statement which should be indelibly inscribed in the memory of every physician and surgeon: "Now focal sepsis is blamed for well nigh every variety of affection. The truth lies between the two extremes." To emphasize the point, the author cites cases throughout the volume showing the danger of drawing conclusions based on incomplete investigations.

It is probably too much to expect every surgeon to study in detail a volume like this, but those who read it and digest its contents will find themselves better practitioners as a result. We consider this book one of the classics of present-day medical literature.

Aids to Surgical Anatomy. By Richard H. Hunter, M.D., M.Ch., Ph.D. New York, William Wood, 1931, 184 pp.

This book is quite up to the standard set by the other volumes of the Students' Aids Series and may be recommended for a rapid survey preparatory to examination.

Modern Proctology. By Marion C. Pruitt, M.D., L.R.C.P., S. (Ed.), F.A.C.S. St. Louis, Mosby, 1931, 404 pp.

The author, like most proctologists, feels that the practitioner is inclined to treat diseases of the anus, anal canal, and rectum too lightly, and he has in a little over four hundred pages set forth the details he considers necessary for them to handle these cases properly.

Of course, the book is too small and superficial for the specialist. For the general practitioner, it covers the ground fairly well.

To one not having access to the larger and more complete works on proctology, this work will be found valuable for quick reference.

Fractures of the Jaws. By Robert H. Ivy, M.D., D.D.S., F.A.C.S., and Lawrence Curtis, A.B., M.D., D.D.S. 180 pp. Phila., Lea & Febiger, 1931.

This is a comprehensive monograph that wastes no words but takes one point and covers its subject thoroughly and plainly. The wide experience of the authors is shown in every page and the illustrations leave little to be desired. For dentists, in particular, this monograph will be of great value.

Maligne Pharynx und Larynxtumoren (Malignant Tumors of the Pharynx and Larynx). By A. Zuppinger. With a foreword by Prof. Dr. H. R. Schinz. 188 pp., with 33 illus. in the text and 9 tables. Leipzig, Georg Thieme, 1931. This is Supplementary Volume 40 of the Fortschritte auf dem Gebiete der Röntgenstrahalen, ed. by Prof. Dr. R. Grashey.

First, the author discusses the normal anatomy and histology of the larynx and pharynx, a knowledge of both being requisite for the proper management of tumors of these organs. Biopsy is of great importance since upon the histological characteristics depends the plan of radiation therapy. Of 257 pharyngeal tumors, 12.4 per cent involved the epipharynx; 40.9 per cent the mesopharynx; and 46.7 per cent the hypopharynx. Only 23 cases of tumors of the interior of the larynx were observed. Of the total of 280 tumors studied there were 20 cases of sarcoma: 10 of the epipharynx, 8 of the mesopharynx and 1 each in the hypopharynx and larynx. Of the 280 cases 68 were still in an operable stage. There were 82 cases without metastases. The treatment was surgical in the operable cases, combined in some with radiotherapy; the remainder were treated with radiation alone.

Eleven cases are symptom-free from fifteen months to nine years. Seventeen cases were symptom-free but have developed recurrences. Fifty cases, although not tumor free, were favorably influenced; that is, there was an improvement lasting more than three months. In 120 cases there was no favorable effect. Thirteen cases were not treated. The divided-dose, prolonged treatment method after Coutard is recommended by the authors.

A lengthy bibliography is appended.

The whole presentation is an excellent example of German thoroughness in presenting full details without hesitating to publish the bad as well as the good results. A series of exquisitely printed microphotographs terminates the work.

Artificial Sunlight, Combining Radiation for Health with Light for Vision. By M. Luckiesh, D. Sc., Director, Lighting Research Laboratory, Edison Lamp Works—National Lamp Works, General Electric Company, Nela Park, Cleveland, Ohio. Pp. 254. New York, D. Van Nostrand Company, Inc., 1930.

The author submits that besides the field of radiation therapy in the cure of illness, which should be the exclusive territory of the medical profession, there is the important aspect of health-maintenance to which it is the responsibility of every art, science, and individual, able to do so, to contribute.

An important point emphasized by the author relates to the folly of non-luminous radiators for heating bodily tissues at a depth. Water is practically opaque to infra-red between a wave length of 14,000 and 80,000 Angström units. Such energy cannot penetrate bodily tissue. Water becomes transparent again to radiant energy between 80,000 and 500,000 Angström units, but the temperature necessary for such radiation can scarcely be used on the human body.

This work is an excellent and extremely useful treatise on the effects of radiant energy, coordinated with the physics of the subject in a manner calculated to be helpful to physiologists, biologists, and the medical profession, as well as to the physicists, engineers and others interested in lighting problems. The viewpoint of the book is particularly directed toward the health-maintaining value of radiant energy in addition to lighting for vision.

Lehrbuch der Röntgenstereoskopie (A Text-Book of Roentgenstereoscopy). By Dr. Max Cohn, Director of the Roentgen Department of the Stadt Krankenhaus in Friedrichshain, Berlin, and Dr. Walther

Barth, Physicist to the Agfa-Filmfabrik, Leipzig. With 193 illus. in the text and 2 tables of detachable stereoroentgenograms. Pp. 323. Leipzig, Georg Thieme, 1931.

In view of the growing employment of stereoscopy in roentgenography, the authors determined to place in print an authoritative account of the physical basis of stereoscopic vision; an historical survey, in which they refer to Case's early efforts in stereoroentgenography of the alimentary tract, and a very complete description of the apparatus and methods of application of the stereo principle to practically every phase of roentgen diagnosis. There is a lengthy discussion of stereofluoroscopy, with due reference to the pioneer work of Caldwell. It is probably no fault of the authors that they fail to note the success attained by the Medical Department of the United States Army during 1918 in producing a practical stereofluoroscope, for apparently the work in this field was not published. The perfection of the Bucky diaphragm by Hollis Potter has so improved the detail in roentgenograms, which otherwise would hardly be satisfactory as single films but very nice stereoscopically, that some of the need for stereo vision has faded away. However, there is still abundant room for much stereoroentgenography. The book constitutes an encyclopedia of information on the subject, especially if one desires to use the method for accurate third dimension measurements.

Cutaneous X-ray and Radium Therapy. By Henry H. Hazen, M.D., Professor of Dermatology, Medical Department of Georgetown University; Professor of Dermatology, Medical Department of Howard University; Member of American Dermatological Association, American Roentgen Ray Society. 160 pp., profusely illustrated. St. Louis, Mosby Co., 1931.

The author's effort to write a short, essentially practical book which would aid in the selection of cases for irradiation, has been realized in the text before us. Sane and safe advice is given as to technique, and many pitfalls are warned against. Special warning is given against the dangers of an excessive number of small doses. Following a brief history of the development of roentgen and radium therapy, there is a practical chapter on the estimation of dosage. We agree with most of the statements made, but physicists will find fault with the statement that the quantity of radiation varies as the square of the voltage. This is not quite true, but there are easily available curves of the intensity rise as the voltage increases, with which the radiologist may orient himself. The author states that stabilizers are not popular among dermatologists; we strongly recommend that they become familiar with the advantages afforded by such device, even in superficial therapy. Since the text of the work was prepared (evidently 1929) a very reliable ionization machine has been devised and is now being widely used, a portable affair, which the reviewer believes every roentgen therapist should employ in the measurement of dosage. We thoroughly agree with the author's insistence upon keeping proper accurate records, and the statement that idiosyncrasy plays a very unimportant part in the production of undue reactions. Some valuable tables, based upon MacKee's formula, are given for unfiltered as well as filtered radiation. The diseases of the skin amenable to radiation, those resistant, and those best treated by radiation combined with some other forms of treatment, are tabulated.

Normale und Pathologische Funktionen der Verdauungsorgane im Rontgenbild (Roentgenology of the Normal and Pathological Functions of the Digestive Organs). By Dr. Rudolf Becker, from the Röntgeninstitut of the Theresienkrankenhaus, Mannheim, and Dr. Albert Oppenheimer, Roentgen Section of the University Polyclinic in Frankfort. 144 pp. 255 illus. Leipzig, Georg Thieme, 1931.

This is a very timely and extremely valuable book, the outgrowth of work done at the University of Frankfort. The peristaltic movements of the entire alimentary tract, including the act of swallowing, peristalsis of the esophagus, stomach, small and large intestine, and the physiological and pathological contraction movements of the gall bladder are all included. The section on the colon is extremely interesting and one which ought to be read by every physician who wishes to understand the physiological phenomena relating to intestinal stasis. Many of the clinical puzzles could be solved were the facts so elaborately set forth

in this work understood by the average clinician. This refers particularly to the explanation of cecal stasis. One fault may be found with this work, as with many of the German works in radiology; that the efforts of American colleagues seem to be almost entirely ignored, if we may judge by the references to American work which we find in this book.

ENCEPHALITIS LETHARGICA, ITS SEQUELAE AND TREATMENT. By Constantin von Economo. Trans. and Adapted by K. O. Newman, M.D. London, Oxford Univ. Press, 1931, 200 pp.

This translation of a classic monograph is well done. The author, the translator and the publisher are to be congratulated for making this valuable treatise available to the English reading physician.

For those interested in encephalitis lethargica, this book is indispensable though the average practitioner will probably find the subject adequately covered for his use in the textbooks and systems of medicine. The recent death of Professor Economo lends a special interest to the publication of this translation.

THE INFANT WELFARE MOVEMENT IN THE EIGHTEENTH CENTURY. By Ernest Caulfield, M.S., M.D., with a Foreword by George Frederick Still, M.A., M.D., (Cantab.), Hon. LL.D., (Edin.), F.R.C.P. (London). 8 illus. N. Y., Paul B. Hoeber, Inc., 1931.

Propaganda, having developed into the high art and the mighty one that it is today, has easily led most of us to the assumption that the infant welfare movement is an element of human progress that belongs rather strictly to the twentieth century. A few interested students, however, have taken the trouble to penetrate the fog of modern ballyhoo and have pointed out abundant instances of concerted efforts toward improving the estate of children that occurred in earlier and darker centuries of the Christian Era. G. H. Payne, for example, has written an admirable narration of the growth of foundling hospitals on the continent, these dating back to that established by Datheus, Archbishop of Milan, in 787. Forsyth, Ruhräh, G. F. Still (who has contributed the foreword to Dr. Caulfield's book) and others have likewise dispelled from the minds of their readers the idea that child welfare never entered the public conscience or consciousness until our own omniscient and compassionate age.

Dr. Caulfield, in this present volume, presents a most vivid and comprehensive picture of some very notable advances that took place in England, more especially in London, in the 18th century.

These advances had little to do with the refined sort of knowledge or method that characterizes the modern science of pediatrics. They were related more closely to a public awakening and a public realization of the value of the life of a child; a change in the public attitude toward the welfare of infants and children. They resulted very largely from the sustained and devoted efforts of a few men with hard minds and soft hearts, men like William Cadogan, Jonas Hanway, Thomas Coram and George Armstrong.

The lot of the child of the early part of the 18th century in England was a miserable one indeed. Infanticide was rife. Neglect, indifference and cruelty were the order. Even among the "quality," children were commonly looked upon as a nuisance and were given over to the care of wet nurses and servants, both because of the fashion of the day and because of the low estimate of a child's value. The mortality below the age of two was about 75 per cent among the general population of children. Among foundlings it was above 90 per cent. The earlier laws and earlier foundling institutions were seized upon by public officials and all manner of low persons as opportunities for exercising diverse chicaneries through which the sorry state of children could be capitalized.

Scenes of this tragic state have been painted for us, dramatically by Dickens, satirically by Swift, and realistically by Henry Fielding, from whom Caulfield has drawn for some of his material. He has drawn as well and with fine discrimination and masterly interpretation from a wealth of literature of that day.

His sketches of the leaders in the infant welfare movements of this period are crisp and informing, that of Jonas Hanway attaining some fine romantic touches. His narration of how these men founded institutions, passed legislation and influenced the public attitude for the welfare of children and transplanted our views on the rearing of children from the realm

of "old wives" traditions to that of scientific observation makes an absorbing story. In their cumbersome style these men were trying to preach what George Ade has epitomized in his own inimitable version: "The hand that rocks the cradle should be amputated."

The illustrations, especially the portraits of Coram, Cadogan and Hanway add measurably to this valuable little book.

THE DIAGNOSIS AND TREATMENT OF VENEREAL DISEASES IN GENERAL PRACTICE. By L. W. Harrison, P.S.O., M.B., CH.B., F.R.C.P.E. Director of Venereal Department, St. Thomas's Hospital. With a chapter on Medico Legal Aspects of Venereal Disease, by F. G. Crookshank, M.D., F.R.C.P. Ed. 4, Oxford Univ. Press, 1931.

That this most excellent text on venereal diseases has been found exceedingly well suited to the needs of the general practitioner is attested by the recent appearance of the fourth edition. An examination of the book revesal ample reason for its popularity. Its matter is limited quite strictly to the practical problems that confront the general practitioner, and the space division corresponds very nicely with the relative importance of the various subjects as met with by the modern physician in general practice.

The arrangement will appear somewhat unusual to those who are accustomed to the American texts. Few American texts, for example, devote so much space to differential diagnosis, or cover such a wide scope of practice. The later American texts, instead of covering all the venereal diseases, and in both sexes, are more apt to be limited to either gonorrhea or syphilis, and if on gonorrhea to concern only the condition in one of the sexes.

The sections on laboratory procedures and interpretations will be found especially helpful to those who are so situated as to be obliged to do their own laboratory work.

The section on the treatment of syphilis is especially comprehensive and includes all the more modern methods of attack on neurosyphilis.

The author exhibits a skillful style that combines clarity and force with a commendable economy of words.

The final chapter, on the medico-legal relations of venereal diseases, by G. F. Crookshank, is somewhat involved, largely because of the perhaps unavoidable use of legal terminology and phraseology. The matter here holds in the broadest principles only in America, since it is treated with the thought of British law in mind. In the other chapters as well, there are occasional references to drugs and methods with which Americans are quite unfamiliar.

The colored plates deserve the highest praise and add measurably to the text.

PRINCIPLES OF PREOPERATIVE & POSTOPERATIVE TREATMENT

REGINALD A. CUTTING, M.A.,PH.D.,M.D.,C.M.

TWELFTH INSTALLMENT

CONTENTS

[This Number]

Chapter		Page
XXI.	Immediate Treatment of Extensive Superficial Burns	705
XXII.	Treatment of Acute Traumatic Craniocerebral Injuries	725
XXIII.	Care of Patients with Intestinal Obstruction	761

[Previously Issued: January to September 1931]

		Volume XI
Introduction		147
I.	General Principles Underlying Rational Preoperative Treatment	159
II.	The Preoperative Treatment of the Average "Good Risk" Surgical Patient	199
III.	The General Postoperative Care of the Average "Good Risk" Laparotomy Patient	377
IV.	Shock and Collapse	413
V.	Blood Transfusion	577
		Volume XII
VI.	Water Balance, Dehydration, and the Preoperative and Postoperative Administrations of Fluids	167
VII.	Disturbances of Acid-Base Equilibrium: Acidosis and Alkalosis	345
VIII.	Abdominal Distention and Gas Pains	563
IX.	Dilatation of the Stomach	597
		Volume XIII
X.	Urinary Disturbances	133
XI.	Postoperative Complications	165
XII.	The Treatment of Operative Wounds	335
XIII.	Postoperative Peritonitis	397
XIV.	Thrombophlebitis	591
XV.	Miscellaneous Postoperative Complications	609
		Volume XIV
XVI.	Care of Patients with Prostatic Hypertrophy	509
XVII.	Care of Surgical Diabetic Patients	517
XVIII.	Care of the Gall-Bladder Patient	526
XIX.	Care of Patients with Tonic Goiter	545
XX.	Preoperative and Postoperative Care of Gastric Cases	570

CONTENTS OF CHAPTER XXI

A. J. S. PAGE

I. Burns considered merely as local lesions 705
II. The fundamental importance of the associated systemic reaction, toxemia 706
A. Blood concentration 708
B. Chloride retention 709
C. Blood sugar variations, adrenal changes 710
D. Other metabolic and tissue changes 710
E. Clinical manifestations of burn toxemia 711
III. Treatment 712
A. Shock 712
B. Local therapy 714
1. Debridement 714
2. Simple removal of devitalized tissue 715
3. Tanning 716
C. Constitutional treatment 721
1. Blood transfusion 721
2. The value of water and sodium chloride 721
3. The use of adrenalin and dextrose 722
4. Physical therapy 722
References 723

Chapter XXI

THE IMMEDIATE TREATMENT OF EXTENSIVE SUPERFICIAL BURNS

Although not connected with the performance of major surgical procedures, the treatment of cases of burns devolves upon the surgeon, and in view of the relatively high mortality rate occurring in connection with the severer degrees of burn trauma, a consideration of the immediate therapy of these cases, as at present understood, is herewith undertaken.

I. Burns Considered Merely as Local Lesions

Burns, considered apart from their systemic manifestations, represent a type of lesion scarcely to be differentiated from ordinary wounds; they are characterized by a relatively sudden solution in the continuity of surface tissues, caused not by physical violence, as in the case of ordinary wounds, but by energy of a physico-chemical nature, (1) heat, whether moist or dry, (2) certain chemicals, such as strong acids and bases, phenol, iodine, and phosporus, (3) actinic radiant energy, as from the sun or from artificial generators like the quartz lamp, and (4) electricity. All of these agents, and possibly others, are capable of producing lesions similar in all essential details to one another, and not dissimilar to ordinary wounds in the process of repair through which they pass during the healing process.

Considered as local lesions, burns differ from ordinary wounds mainly in the fact that they tend to involve large surface areas and consequently, when extensive and deep, to eventuate in excessive, disfiguring, and deforming scar-tissue formation. The time-worn classification of burns into three or more "degrees" seems to serve no really useful function except as it tends to focus the attention of the surgeon in any given case upon the probability of the development of excess scar-tissue formation. A classification of local burn trauma into

two degrees based on the expectancy of scar-tissue formation, (1) superficial, and (2) deep, in accordance with the suggestion of Bancroft and Rogers[1] and others, seems more rational, and may eventually become the accepted nomenclature. Under such a classification superficial burns would include all those which do not eventuate in the complete destruction of the skin epithelium, especially such islands of epithelium, representing hair follicles and coil glands, as lie at a lower level than the stratum germinativum. Obviously, if a sufficient number of such islands remain undestroyed after a burn, they tend to act as natural Reverdin skin grafts from which relatively complete and normal epithelialization may proceed. The deep burns, on the other hand, would be represented by lesions of greater penetration in which epitheliazation could not occur except from the edges of the lesion, all islands of epithelium having been destroyed, and which, consequently, invariably would require artificial skin grafting during the process of repair and possibly other surgical measures aimed at the production of satisfactory functional and cosmetic results.

Aside from this very important practical difference between the lesions of burns and ordinary wounds, there is not very much of therapeutic novelty as far as burn lesions are concerned. Hemorrhage and infection tend to present problems in the one as in the other; especially is this true with respect to infection. The possibility of tetanus and gas bacillus infection should be carefully considered.

II. The Fundamental Importance of the Associated Systemic Reaction, Toxemia

The real problem presented by extensive burns is not connected with the local lesion as such, but rather with certain concomitant systemic reactions of toxic nature which in themselves may be sufficiently severe to jeopardize the patient's life.

The severity, as concerns life, of a case of burns depends not so much upon the depth of the trauma or the gross amount of

tissue actually destroyed as upon the *extent* and *location* of the skin area involved. Living skin, when subjected to the trauma known as burning, is capable of developing certain complex chemical by-products, the exact nature of which is unknown, but which are absorbable, and which when absorbed into the circulation produce systemic reactions described collectively as "toxemia." In this respect skin differs from other tissues; the burning of fat, muscle, osseous, and other tissues is accompanied either by a very minor degree of such a reaction or none at all.

The burning of certain particular skin areas seems to be acompanied by the development of severer grades of toxemia than certain other areas of similar extent. This is particularly true of the so-called "blush area," which, as the term implies, comprises the face, neck, and upper torso, especially the anterior part of the chest wall. The rich blood supply of this area and particularly its sensitive vasodilatory capillary system doubtless partly at least accounts for the rapidity and degree of toxic absorption.

Much of our knowledge of the process of toxic absorption has been developed by the researches of Robertson and Boyd.[2] Characteristically, toxemia develops only when living skin is burned, as is shown by the fact that extracts from burned dead skin, when injected into experimental animals, fail to produce typical toxemia, whereas, if burned living skin be engrafted upon an otherwise normal animal a characteristic toxemia develops.

There is apparently a definite latent period of about eight hours between the occurrence of a burn and the beginning of toxic absorption, since it has been shown experimentally that if complete excision of a burned skin area be performed within such a period, the toxemic constitutional reaction does not develop, whereas if excision be delayed beyond this latent period, the procedure of excision is without avail. The eight hour latent period of toxic absorption, as just described, represents not the interval beyond the limits of which clinical

symptoms of toxemia may be expected to appear, but only the period beyond which the surgical eradication of the local lesion can no longer be depended upon to prevent the subsequent development of toxemia. The clinical symptoms of toxemia do not ordinarily appear for from twenty-four to forty-eight hours, or even longer, after the infliction of a burn.

That the development of burn toxemia depends upon absorption of toxin by way of the blood stream is apparently demonstrated by the fact that the development of such a toxemia does not take place if the vascular drainage from the involved area be interrupted by ligation of afferent blood vessels. Furthermore, evidence has been adduced to show that the absorbed toxin is carried mainly either within or absorbed to the red blood cells, since, whereas whole blood derived from burned animals, when injected into normal animals is toxic for them; blood serum, on the other hand, is found not to be toxic except in relatively large doses.

The fundamental nature of burn toxemia is very imperfectly understood, but certain of its clinical and post-mortem changes have been investigated with considerable care, and not a little information has been gained thereby.

A. Blood Concentration: The development of burn toxemia is accompanied by a very definite concentration of the blood in the more severe cases, and this concentration has been found clinically to vary directly with the severity of the burn.

Blood concentration is most conveniently estimated clinically by means of hemoglobin determinations; Underhill[3] has reported clinical increases to 145 per cent and Ravdin[4] to 125 per cent of normal. This blood concentration in the more serious cases is capable in itself of causing death, because such degrees of viscosity lead to impairment of capillary circulation, diminished oxygen-carrying capacity of the blood, lowering of temperature, suppression of urinary secretion, and suspension of the vital activities.

Locke[5] analyzed the erythrocyte count in 9 cases of severe burns, and found that in 5 cases in which the count exceeded

9,000,000 to the cubic millimeter all the patients died, whereas the other 4 patients, in none of whom did the count exceed 7,260,000, recovered. Similarly, Underhill,[3] studying 21 clinical cases of burns, found that in cases regarded as "serious" the hemoglobin content was always more than 125 per cent of normal. This author reports 1 exceptional case in which the hemoglobin content reached the astounding figure of 209 per cent. Pack[6] believes that the maintenance of a 40 per cent increase in the hemoglobin is inconsistent with life.

B. Chloride Retention: According to the researches of Underhill, which have been amply confirmed by other investigators, the blood chloride content of the blood in burns is characteristically considerably reduced. The reduction in severe cases may be of the order of 50 per cent; figures of 250 mg. or even 200 mg. of chlorides to the 100 c.c. of plasma are not of very infrequent occurrence. This reduction of the blood chlorides is not accompanied by a corresponding urinary chloride excretion; in fact, the very opposite effect occurs, the urine showing a markedly decreased chloride output. Only one explanation for such a state of affairs is evident, the occurrence of "chloride retention"; furthermore, the chlorine radical, not being present in normal amounts either in the blood or the urine, must have been stored in some of the fixed tissues of the body. It is generally assumed that such storage represents a conservative process in which the chlorine radical combines with the primary toxic materials elaborated in the burned tissues and subsequently absorbed into the blood stream in such a manner as to render them innocuous. This theory corresponds with the observed facts in that excessive chloride excretion occurs at the end of the toxemic period, i.e., at the time of the separation of sloughs. In lobar pneumonia is found a close analogy in that during the height of the disease there is marked chloride retention, but after the crisis excess chloride excretion.

Davidson[7] has shown that the lowering of the plasma chlorides is proportionate to the amount of tissue devitalized and

persists as long as sloughs remain. Possibly the disappearance of the chlorides from the blood stream accounts at least in part for the blood concentration observed clinically, since a large part of the osmotic tension of the blood stream is dependent upon its chloride content, 60 per cent according to Howell.[8] Barach[9] has determined that the intravenous injection of 100 c.c. of 15 per cent sodium chloride solution is capable of increasing the total blood volume by 500 to 1200 c.c.

c. Blood Sugar Variations, Adrenal Changes: At autopsy on patients who have died from extensive burns the adrenal glands are found to be from three to five times normal size, and both the epinephrine content of the medulla and the lipoid content of the cortex are markedly decreased (Weiskotten[10] Olbrycht[11]). On microscopic section the glands show definite degenerative changes.

Blood sugar estimations on specimens of blood taken from such cases during the agonal period may be very low. Greenwald and Eliasberg[12] performed a series of experiments on rabbits which indicate that early in the development of cases of burns (one and a half hours after the infliction of such a lesion) the blood sugar concentration may be very high, a phenomenon apparently due to excess stimulation of the adrenal medulla, since the medullary tissue microscopically shows evidence of excessive activity. This occurs at a time previous to the development of toxemia as determined both clinically and experimentally, and therefore, must represent no essential part of toxemia as such; the phenomenon seems to be closely related to the severity of shock. Later, however, microscopic changes in the medulla are found, suggestive of exhaustion, and during this period in severe cases the blood sugar is found to be correspondingly low; these changes occur primarily in the period, clinically and experimentally, characterized by toxemia.

d. Other Metabolic and Tissues Changes: Additional tissue and metabolic changes have been noted, which, so far as is now known, are of relatively minor importance. Degenera-

tive lesions have been described at different times in various parenchymatous organs, liver, spleen, pancreas, and other tissues. Most pathologists regard these as largely adventitious. The occurrence of ulceration in the gastrointestinal tract as a complication of burns i.e., the so-called Curling's ulcer is probably less frequent than usually believed. Novak[13] estimates the incidence in fatal cases at 5 per cent. Ronchese[14] saw only 1 case in 348 autopsies. Ecchymotic patches on the small intestine, both subserous and submucous, are commonly found, at least experimentally.

Apparently changes in the acid-base balance of the body are not characteristic of burn toxemia as such; they may occur as more or less accidental complications of blood concentration or excess chloride administration. The same applies to disturbances in protein metabolism; whereas increases in the non-protein and urea nitrogen of the blood do characteristically occur, they are usually not particularly significant, and can probably be explained in most or all cases solely on the basis of blood concentration. Albumin and casts are more or less constantly demonstrable in the urine of burn cases, but appear to be solely dependent upon blood concentration; the kidney parenchyma is ordinarily not demonstrably damaged.

E. Clinical Manifestations of Burn Toxemia: As previously mentioned, the clinical manifestations of toxic absorption usually appear only after twenty-four to forty-eight hours. They consist of:

(1) Elevation of temperature
(2) Rapid pulse
(3) Vomiting
(4) Drowsiness or delirium

The development of such symptoms and the development of the previously described underlying pathology may be expected to occur in all cases of excessive burns, even though very superficial. In this respect, the real seriousness of burns is apt to bear no particular relation to the problem presented by the actual local lesion.

Pack[15] considers as serious all burns deeper than first degree which involve one-tenth or more of the body surface. Burns deeper than first degree which involve more than one-third of the body surface may result fatally, and those involving more than two-thirds usually do result fatally in adults. Much less extensive burns in children often result fatally.

The estimation of the severity of burns in accordance with their extent relative to the total body surface is, of course, a rather inaccurate method, but one which suffices well enough for determining the severity in ordinary clinical cases. In any actual estimation some fairly accurate method of judging the surface extent of various component parts of the body becomes desirable.

Berkow[16] has estimated the distribution of body surface area as follows:

	Per Cent
Lower extremities (including the buttocks)	38
Trunk (including neck)	38
Upper extremities	18
Head	6
	100

The hand accounts for one-fourth of the surface of the upper extremity. The foot accounts for one-sixth, the leg one-third, and the thigh one-half of the surface of the lower extremity. Estimations based on such a schema as this seem to present rather obvious advantages over mere random judgments.

III. Treatment

The treatment of cases of burns may be artificially divided into two parts, (1) the treatment of the local lesion, and (2) the treatment of the systemic reaction; but theoretically and actually such a division is not a convenient one, because the degree of toxic absorption depends very largely upon the efficiency of the treatment accorded the local burned area.

A. Shock: In the early stages of burns, i.e., before the end of eight hours, toxic absorption has not yet occurred, and the

gravity of the patients's condition is largely dependent upon the degree of shock present. The condition of shock occurs frequently in connection with burns, especially those of a more severe nature, and unless the condition be recognized and accorded successful preliminary treatment, the patient's chances of recovery may at least be jeopardized, and in extreme cases such an oversight may actually prove fatal.

There is a real danger that the general condition of the patient at the time of being first seen will quite escape the attention of the medical attendant in his zeal to do something locally, if he possesses the information that the local wound is the source of the toxemia and is attracted to this area by the silent or expressed entreaties of both the patients and their friends. No undue anxiety need be felt, however, in the usual case of thermal burns to hasten in the matter of local treatment, since a number of hours of grace are allowed by Nature before the beginning of the onset of the other main factor in mortality, "toxemia."

Formal treatment directed toward the relief of local conditions is highly irrational in the presence of shock. The truth of this contention is altogether too rarely appreciated, and it is no uncommon sight in some hospitals to observe the uninitiated, painstakingly laboring with the removal of clothing, puncturing, and cutting away blebs, and washing away contaminating debris, while the patient's systolic blood pressure is less than 70, 60, or even 50 mm. of mercury, and the exposure incident to the manipulation is constantly aiding in the further dissipation of heat from a body which already manifests a subnormal temperature. The shock of hasty and unwise local treatment added to the shock already present as the result of the original burn trauma, undoubtedly precipitates many a needless death.

The treatment of burn shock differs in no essential particulars from the treatment of shock otherwise induced, the essential features being the administration of large doses of morphine, the maintenance of the body warmth by the applica-

tion of external heat in the form of a light tent or hot water bottles and internal heat in the form of warm drinks or intravenous infusion, the lowering of the head to combat cerebral anemia, and the promotion of quiet and rest under the watchful eye of a competent attendant. During this period the local trauma may be completely disregarded except for the prevention, so far as is feasible, of adventitious local contamination; for this purpose the wounds may be simply covered with a sterile sheet or towels. It is again emphasized that toxic absorption does not occur for several hours, a period of time ample both for the treatment of the shock and for the institution of suitable additional treatment after the period of shock has passed.

Two important exceptions to this discussion must be mentioned in passing:

(1) In those cases in which the patient's clothing has caught fire the utmost care must be exercised immediately after the patient is seen to make sure that no smoldering material has been over-looked in the excitement of transporting the patient to the medical advisor.

(2) In attending cases of chemical burns one peculiarity of the traumatizing agent must be remembered; a prolongation of action is maintained until neutralization of the active agent is accomplished, either by natural or artificial means. Patients burned by chemicals should be quickly treated locally to assure complete neutralization of the chemical reagent before anything else is done. The neutralizing property of alcohol in connection with phenol burns can be invoked immediately. In most other cases copious gentle washings with water is indicated, after which application of a neutralizing agent may be employed, weak acids for strong bases, and vice versa.[17]

B. Local Therapy: 1. *Debridement:* Granted that chemical reagents have been successfully neutralized and shock has been found absent or has been successfully combated, the attention of the medical attendant is, naturally, next focussed on the local lesion. In accordance with what has been developed of the therapy of wounds of other kinds, it would, at first thought, appear rational to perform complete debridement, thus converting the contaminated wound into a clean one. Such a

procedure has, in fact, been warmly advocated by Brager, Lewis, MacKenzie, and others.[18] For the purpose of debridement the patient is placed under a general anesthetic, and after the patient's clothing over and surrounding the burned area has been cut away, or otherwise carefully removed, the latter area is either thoroughly scrubbed with a sterile scrubbing-brush and sterile water or saline solution until the area is completely clean, or else resort is had to various sharp instruments for the removal of the contaminated tissue, amputation knives, razors, curettes, and scissors,

2. *Simple Removal of Devitalized Tissue:* Whereas such heroic therapy is undoubtedly the treatment of choice in connection with the deep charring of a limb or in cases of other deep burns of limited extent, one of the usual characteristics of burn trauma is that it is extensive, though superficial, and one of the complications most to be feared is the subsequent formation of excessive scar formation with its train of contractures and series of plastic repairs. So important is this matter that it is usually considered not only proper, but necessary, to preserve as much of the undamaged epithelial covering of the traumatized area as possible, from which epithelial proliferation may subsequently proceed, in order that scarring may be reduced to a minimum. Accordingly it seems best to make an exception of burns in the matter of debridement and to be content with a macroscopic cleansing of the burned area without attempting the removal of any healthy tissue that may subsequently be utilized by Nature in the process of repair.

Frequently, in the severe type of cases, the patient may be placed in a bathtub filled with warm water and the clothing gently soaked and cut away; in cases less severe it may be possible by gentle manipulation to dispense with any such formal procedure and attack may be launched upon the traumatized area directly, loose skin being stripped away with sterile forceps, particles of dirt being carefully removed likewise, blebs being punctured and cut away with scissors and the

entire area being finally thoroughly cleansed with some non-irritating fluid like normal saline solution or benzine, the latter substance being particularly grateful as far as the patient's sensations are concerned, and at the same time being somewhat antiseptic; in the very severe cases, on the other hand, the proper performance of the preliminary cleansing may not be attempted in the absence of general anesthesia, and in such cases no hesitation should be felt in taking the patient directly to the operating room and making the process as formal as may be necessary to ensure thoroughness.

3. *Tanning:* Undoubtedly the toilet of the local lesion serves as a valuable prophylactic precaution against the development of subsequent toxemia, but inasmuch as much material of toxic nature is, of necessity left behind, some method of further preventing absorption is desirable and this, fortunately, has been provided in the process of chemical fixation of this material *in situ*. Various substances have been suggested for this purpose, amongst which may be mentioned absolute alcohol, picric acid, and "aluminium aniline," but since 1925 the method of tannic acid fixation has become virtually standard to the exclusion of all others. This method was first promulgated by Davidson[19] and seems most completely to meet the indications. In this method a true tanning process fundamentally similar to that used in the preparation of leather is employed.

A watery solution of tannic acid of a strength between 1 and 5 per cent has been advocated; possibly the best results follow the use of a 2 or 2.5 per cent solution. Tannic acid keeps indefinitely in powder form, but in solution fairly rapidly changes into gallic acid, which is unsuitable for the purpose now being described, and consequently solutions of tannic acid should be freshly prepared as needed. Inasmuch as tannic acid solutions are antiseptic by virtue of their protein coagulating properties they require no preliminary sterilization, though it is well to take the precaution of using sterile water in the preparation of the solution in order to avoid gross contamination.

The application of tannic acid solution to the traumatized area may be made either by means of wet gauze compresses or by spraying the solution directly on the area, according to the

FIGS. 64 and 65. Two views of a patient being treated for superficial burns of chest and abdomen by tannic acid method; area has been thoroughly tanned and crust partly dried.

FIG. 64. A close-up; note expression of comfort on patient's face. Demarcation line between tannic acid crust and normal skin is sharp, showing that tannic acid has no effect on vital tissue.

choice of the operator or the facilities at hand. An atomizer of the type commonly used for spraying the nose and throat makes a very suitable spraying device, and almost any available sterilized gauze squares may be used for making compresses: squares about 10 to 12 in. on a side and 8 to 10 layers in thickness when loosely wrung out of the solution and spread over the burned area make excellent compresses.

In the application of the solution by either method the aim is to keep the burned surface continually moist with the solution. The solution evaporates from the skin when applied by means of an atomizer in about twenty minutes, and gauze

compresses become unduly dry in about half an hour; consequently, renewed application of solution is required at the end of these respective time intervals.

FIG. 65. Patient went to sleep during photography. A wire heat-tent is in place; note extension cord and electric light socket fastened to tent by adhesive plaster. A simple arrangement of this kind is all that is required. The bedclothing will be draped over the heat-tent.

The electric light tent serves as an excellent device for holding the bedclothing away from the patient, thus facilitating the manipulations incident to the application of tannic acid. It is no less valuable in relieving the pressure of bedclothing during the preliminary treatment of shock or yet again during the process of drying the tanned area, immediately to be described; in both of the latter mentioned stages of treatment, in addition to acting as a support for the relief of pressure, it also serves for the application of heat.

Tannic acid unites with devitalized tissues to form a complex protein-tannate, but normal tissues resist this action, and

for this reason the action of tannic acid on the normal margins of burned areas presents no problem.

Tanning of burned areas is usually complete in from sixteen to twenty-four hours, the method of spraying being somewhat more rapid in its action than the compress method. When tanning is complete the traumatized area presents a uniform mahogany-brown color and is, of course, thoroughly moist; indeed, when the compress method is used a thorough moistening of gauze packs should precede their final removal lest portions of tissue which have become partially adherent through drying be inadvertently torn away with the gauze.

During the process of tanning the toxic products caused by the burn trauma are partly imprisoned within the tannate coagulum and toxic absorption is thereby lessened; however, cessation of absorption occurs only when the tanned area has been subjected to a process of thorough drying; and for this reason desiccation should be instituted without delay as soon as the full action of the tannic acid has been realized. Drying is most conveniently achieved by utilizing the electric light tent and keeping the temperature beneath the tent at 100° F. or slightly more, the patient himself being naked at least as far as the involved area is concerned. Under these conditions drying is complete in twenty-four hours or somewhat longer, depending to a considerable extent upon the depth of the burn being treated. When dry, the tanned area presents a hard, leathery, dark brown or black crust or splint fashioned out of the patient's own tissues (Figs. 64 and 65). This crust performs three important functions apart from the prevention of toxic absorption:

(1) It protects sensitive nerve ends from extremes of temperature and mechanical stimulation, thereby ensuring to the patient relief from pain. Patients lie on the crust in perfect comfort.

(2) It interposes an impervious barrier between tissue spaces and injured tissue cells, and the external air thereby preventing evaporation of tissue fluids and exudates. This is important in connection with the dehydration found characteristically in burn toxemia.

(3) It serves as a protective covering for young granulation tissue and new epithelium during the process of repair.

On the other hand, the crust may occasionally become objectionable due to the formation of pus below its surface. No method, with the single exception of rigid debridement, can entirely prevent the development of suppuration, and the tannic acid treatment, therefore, simply shows this limitation in common with other methods; however, when suppuration does occur under tannic acid therapy, it does so comparatively late as far as the period of toxemia is concerned, and no hesitation should be felt in cutting away the crust for purposes of drainage. The crust is conveniently softened prior to removal by treating it for a short time with liquid petrolatum; thereafter it can be easily cut with a knife or scissors.

Infection should be conveniently kept at a minimum in the treatment of burns by the use of tannic acid by virtue of the very nature of the method. Tannic acid, as previously noted, is, to a degree, antiseptic, and unless grossly infected material be covered over during the process of tanning widespread suppuration should not ordinarily occur. In applying the method the ordinary surgical principles with regard to "contamination" and "infection" should be borne in mind. For a period of eight hours or so following any trauma organisms which may have found their way to the traumatized area do not ordinarily penetrate tissues to any considerable extent. This is because they have not yet had time to excite sufficient tissue reaction to call forth the characteristic leucocytic response in accordance with which they become engulfed in the actively motile polymorphonuclear cells and transported deeply into the tissue spaces and the local lymph stream. Accordingly, if burns are seen and treated early, virtual sterilization of the traumatized area should be the rule.

A rather homely but practical emergency suggestion has been offered in connection with the tannic acid treatment of burns by Shen.[20] If tannic acid, as such, be not readily available an infusion of tea serves almost as well; about 8 gm. of tea per

100 c.c. of water when boiled for fifteen minutes make a suitably strong solution of tannic acid.

The many other methods of local treatment in burn trauma which have been described from time to time in the past are of value in direct degree as they fulfil the main indications so admirably met by the tannic acid treatment, (1) prevention of toxic absorption, (2) prevention of infection, (3) prevention of pain, (4) prevention of dehydration, and (5) protection from trauma. None of them will be here described, however, because none of them sufficiently well meet the indications to make them of any considerable value in serious cases.

c. CONSTITUTIONAL TREATMENT: Theoretically the fixation by coagulation of toxins in the traumatized area in burn cases should prevent the subsequent development of toxemia, and systemic treatment should not, therefore, be required except for primary shock and hemorrhage. Unfortunately, this ideal condition is never achieved practically. Many burn cases are seen too late for the adequate prophylactic treatment of toxic absorption; those which are seen early and are properly treated do not respond according to theoretical consideration, and complications, especially infection, frequently induce profound systemic effects. For these reasons the systemic treatment of burns generally bulks large.

1. *Blood Transfusion:* The therapeutic possibilities of blood transfusion should always be kept in mind not only in the early stages of shock but also later in connection with the development of toxemia and sepsis.

Davidson[21] transfuses all patients who have received a possibly fatal burn, and in all cases which develop shock the transfusion is repeated.

The heroic "exsanguination transfusion" of Robertson and Boyd[22,23] may occasionally save a life but is rather radical therapy in view of its unproved value.

2. *The Value of Water and Sodium Chloride:* The administration of water and sodium chloride in comparatively large quantities constitutes the surgeon's sheet anchor in the pres-

ence of actual or anticipated toxemia; 4000 to 5000 c.c. of fluid in the course of twenty-four hours, whether by mouth, proctoclysis, hypodermoclysis, or infusion, is an amount none too little to administer to an adult in a state of threatened or developing burn toxemia. Sodium chloride is conveniently added to proctoclysis, hypodermoclysis, or infusions in such proportions as to make an isotonic or even slightly hypertonic solution (up to 2 per cent). Sodium chloride is, of course, poorly tolerated *per os*.

The adequacy of the treatment can be determined solely by laboratory tests; hemoglobin estimations or total erythrocyte counts are urgently demanded every three to four hours in severe cases undergoing active treatment, and plasma chloride determinations every six to eight hours are also indicated in these cases. Unsatisfactory reduction in abnormal values demands immediate reduplication of energy in the appropriate direction, either administration by one or more additional avenues of water and sodium chloride, or in the case of the latter substance, increase in concentration.

3. *The Use of Adrenalin and Dextrose:* In accordance with experimental considerations blood sugar determinations should be made in conjunction with chloride determinations, and when unduly low sugar values are encountered either adrenalin or dextrose should be supplied. The exhibition of adrenalin has not been proved to be of clinical value, and it is a relatively dangerous drug when incautiously handled. There seems to be no good reason, however, for withholding dextrose, and every surgical principle seems to indicate the maintenance of the blood sugar level at a normal figure by the adequate exhibition of this substance.

4. *Physical Therapy:* It is quite possible that the light generated in an electric light tent may have some value *per se* in the treatment of burns. Kessler[24] has reported excellent results following the application of light from the ultraviolet and radiant heat lamps in the absence of other treatment, and although such therapy alone cannot be regarded as adequate

for severe burns or even burns of less severity the possible value of actinotherapy as an adjunct to treatment by other methods is strongly suggested. Ultraviolet light is advocated because of its supposed value in the prevention of scar tissue formation and the radiant heat lamp for its penetrating action and stimulating effect on the circulation. The ultraviolet lamp itself, however, when handled indiscretely is capable of causing severe burns. In general, it may be said that the entire subject of phototherapy needs to be placed upon a more secure scientific basis before it can receive the measure of endorsement to which it is probably entitled.

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Chapter XXII

THE TREATMENT OF ACUTE TRAUMATIC CRANIOCEREBRAL INJURIES

CONTENTS OF CHAPTER XXII

A. J. S. PAGE

I. The relative importance of injuries to the contents of the skull and the relative unimportance of skull fractures as such 727
II. The significance of the history in estimating the severity of craniocerebral injuries 728
III. The physical examination 730
IV. Pathological physiology of increased intracranial tension. . . . 731
V. The interpretation of symptoms on the basis of the pathological physiology as outlined. 736
A. Irritative lesions 737
1. Cortical. 737
2. Midbrain. 737
3. Bulbar 737
B. Paralytic lesions 738
1. Cortical. 738
2. Midbrain. 738
3. Bulbar 738
VI. The spinal tap as a direct measure of increased intracranial tension 738
VII. The treatment proper 744
A. Shock 744
B. Early treatment after recovery from shock. 746
C. Conditions demanding primary radical surgery. 746
D. The non-surgical treatment of increased intracranial tension 747
1. The use of hypertonic solutions, dehydration therapy. . 747
2. The therapeutic spinal tap 751
3. Indications for a decompression operation. 753
E. Résumé. 754
VIII. Prognosis. 757
References 759

Chapter XXII

THE TREATMENT OF ACUTE TRAUMATIC CRANIOCEREBRAL INJURIES

1. The Relative Importance of Injuries to the Contents of the Skull and the Relative Unimportance of Skull Fractures as Such

Although it is, of course, true that many or, indeed, most cases of head injuries are not nowadays submitted to formal operative procedures as a part of their treatment, such cases fall within the field of general surgery and, depending upon circumstances, may require operative intervention either soon after they are first seen or at varying intervals of time subsequently. Accordingly, it falls to the lot of the surgeon not only to assume care of such of these cases as present immediate indications for surgical procedures but also to observe and to treat such cases as progress, on the one hand, to complete recovery under conservative therapy, or, on the other hand, in spite of such therapy, to a point at which operative intervention becomes desirable or necessary.

Formerly, the degree of injury to the cranium or bony skull was at least tacitly assumed to be a reliable index of the severity of a case of head injury, those cases in which fractures could be demonstrated being considered less favorable than those in which no fracture could be found, and those with severe or extensive fracture being considered more serious than those with fractures of minor degree. As the result, however, of the researches of such investigators as Cushing, Sharpe, Frazier, Sachs, and others, it is now considered a sound surgical principle virtually to disregard the extent of the bony injury, provided such injury does not demand operative interference for fairly obvious mechanical reasons, and to focus the attention on the internal soft parts injuries the extent of which cannot be gauged directly but only indirectly on the

basis of symptoms, signs, and special methods. The preoperative treatment of the traumatic craniocerebral case, therefore, resolves itself in the average instance into an estimation of the nature and extent of the injury to the brain substance, the meninges, and the accompanying blood vessels, and the application of suitable measures for the relief of the same with a view to obviating the necessity for surgical interference or to alleviating the danger incident thereto in case surgery becomes inevitable.

As representative of the trend of modern practice away from operative intervention and toward conservatism, two series of cases may be compared, the first collected by Sharpe,[1] and the second reported by Weaver:[2]

> Sharpe reviewed the histories of skull fracture cases from three of the large hospitals in New York City during the decade, 1900–1910. The immediate mortality from fracture of the skull was between 46 and 64 per cent. Of those cases submitted to operation 86 per cent died; of the 34 per cent of those recovering and discharged as "well," "cured," or "improved" which could be traced, 67 per cent were still suffering from the effects of the former injury.
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> In the series of cases reported by Weaver only 36 cases were submitted to formal operation in a series of 125 consecutive serious head injuries, and only 22 deaths resulted.

II. The Significance of the History in Estimating the Severity of Craniocerebral Injuries

As an aid in estimating the probable extent of cerebral damage the history of the case is often of considerable value. An estimation of the amount of force probably exerted during the reception of the injury is of importance, since, other things being equal, the amount of the force and the extent of cerebral damage are proportionate. Specifically, elicitation of the history should attempt to reveal:

(a) In cases of blows upon the head, the nature of the object with which the patient was struck and the amount of force used,

(b) In cases of gunshot wounds, the kind of shot used, the kind of gun, and the distance from gun to patient,

(c) In cases of automobile, street car, and train injuries, the speed at which the vehicle was travelling and the position of the patient at the time of the accident,

(d) In cases of falls, the probable distance of descent and the position of the patient on alighting.

Various presumptive evidences of the probable extent of trauma should also be considered; whereas loss of consciousness usually indicates a relatively severe degree of trauma, and the longer the period of unconsciousness the more severe the injury,[3] the occurrence of previous attacks of faintness, indicating possible cardiovascular disease, convulsions, indicating possible epilepsy, diabetes, and uremia, should at least suggest the consideration of other possible etiological factors in the causation of loss of consciousness.

In eliciting the history, special attention should be paid to the presence or absence of the so-called "meningeal syndrome," which consists of (a) a period of unconsciousness followed by (b) a period of lucidity, and followed, in turn by (c) a period of deepening coma.

Such a train of symptoms usually indicates injury to the middle meningeal artery, typically the posterior branch, accompanied by the formation of a hematoma between the dura and the skull.

The intermediate lucid interval in cases of laceration of the middle meningeal artery may last for a period of hours or days, depending, of course, upon the size of the vessel injured and the degree of adherence of the dura to the skull in the involved area. The period of unconsciousness is ushered in by headache, vomiting, and drowsiness which gradually deepens into coma; the blood-pressure rises, and a dilated pupil is found on the affected side. The spinal tap characteristically shows only a clear fluid under some increase of tension, the hemorrhage being extradural.

III. The Physical Examination

In the physical examination of a victim of a head injury, before proceeding to any local examination of the head, one should not omit to recognize "shock" if present, since on the one hand, patients may die of shock alone, and, on the other hand, patients subjected to the trauma incident to even minor operative manipulations during the period of shock present a very high mortality rate. Other things being equal, severe shock may be interpreted as indicating severe injury.

The local examination of the head should embrace the following especial considerations:

(1) The nature and extent of lacerated and incised wounds of the scalp with an estimation of their probable depth and probable involvement of the cranium; sometimes the local escape of brain substance or of cerebrospinal fluid will make the latter clear.

(2) Evidence of injuries to the skull and brain not connected with scalp wounds, especially the presence of abnormal swellings and depressions (depressed fracture).

(3) Evidences of fracture of the base of the skull, especially as shown by examinations of:

(a) *eyes:* subconjunctival hemorrhage and ecchymoses about the upper and lower lids, pulsating exophthalmos,

(b) *ears, nose,* and *mouth:* bleeding, escape of brain substance or cerebrospinal fluid, which always indicates basal fracture in the absence of evidences of obvious external trauma,[3]

(c) *mastoid process:* ecchymoses and edema ensuing usually about twenty-four hours after injury (Battle's sign), which indicates local fracture,

(d) *pupils of the eyes:* equality of size, dilatation or contraction, and reaction to light and distance.

(4) Skiagrams, including at least anterior-posterior and lateral views for the purpose of visualizing the position and extent of any possible fractures present, but always bearing in mind that as an aid in the discovery of basal fractures the *x*-ray method is exceedingly untrustworthy.

In the interpretation of physical findings, apart from the simpler signs which are capable of fairly direct explanation much confusion and difficulty will be avoided by attempting to visualize the special pathological physiology involved in head

injuries. The characteristic symptoms and signs are dependent upon compression of the brain substance; occasionally this may result directly from the local inward collapse of a section of the skull, but in by far the greater number of cases the compression is indirect and secondary (1) to intracranial hematoma formation, (2) to reactionary changes in injured brain tissue, the formation of edema, or (3) interference with the normal circulation of cerebrospinal fluid.

IV. Pathological Physiology of Increased Intracranial Tension

The coverings of the brain, except in the very young child, are incapable of any measurable expansion at all, expansion being prevented (1) by the bony protective covering, the cranium, and (2) by the dense and relatively inelastic dura mater which is also inexpansible even in cases in which the bony supportive envelope has been injured. The normal contents of the skull consist, of course, of (a) the brain substance, (b) the blood vessels, arteries, capillaries, veins, and venous sinuses, together with their contents, i.e., venous and arterial blood, and (c) the cerebrospinal fluid contained in the ventricles and subarachnoid spaces.

Encroachments upon the capacity of the skull from any cause increase the intracranial tension, forcing out of the cavity the cerebrospinal fluid, which is under relatively low tension (8 to 10 mm. of mercury), and the venous blood, which is also under relatively low tension, the former through the foramen magnum into the subarachnoid space of the cord, and the latter by the efferent veins into the general circulation. The amount of expansion which the brain tissue can obtain at the expense of these fluid contents of the skull or the amount of space which can be usurped by foreign material, such as blood clot, is strictly limited, however, for, as the fluid contents are displaced, the tension within the cranium progressively increases and eventually either the pressure becomes so high as to prevent the ingress of sufficient arterial blood to maintain

the function of the vital centers, or these centers become paralyzed by mechanical pressure and the patient succumbs.

Thus, during the process of expulsion of the *cerebrospinal fluid* and of the *venous blood* relatively little clinical change occurs in the condition of the patient; when, however, the intracranial pressure becomes sufficiently high to force arterial blood out of the capillaries, arterioles, and arteries, the vital centers begin to suffer acutely as a result of curtailed oxygenation and a compensating mechanism is brought into play which consists of the intake of increasing amounts of air by the lungs and an increase in the arterial tension.

Among the traumatic lesions which are directly capable of producing a restriction of space in the cranial cavity may be mentioned in addition to depressed fractures of the skull:

(1) Extradural hemorrhage, i.e., hemorrhage which occurs between the cranium itself and the dura, as for example, in rupture of the middle meningeal artery and more rarely rupture of the venous sinuses,
(2) Subdural hemorrhage,
(3) Subarachnoid hemorrhage,
(4) Subpial hemorrhage,
(5) Intracerebral, intracerebellar, or intramedullary hemorrhage or edema,
(6) Intraventricular hemorrhage,
(7) Increased secretion or defective absorption of cerebrospinal fluid indirectly due to trauma.

Certain of these lesions, like subdural hematoma and depressed fracture, rather obviously are capable of producing mainly localized pressure, whereas, others like cerebral edema produce mainly diffuse pressure.

As a result of trauma to the brain, its tissue, like other tissue, undergoes certain reactive changes, among which is swelling, due to outpouring of serum and cells into the tissue spaces of the injured area; the swelling of brain tissue, however, as previously mentioned, can take place only at the expense of fluid contents capable of displacement, since increase of the

cubical contents of the cranium is impossible, and consequently any injury to brain substance, whether or not primarily localized, also becomes ultimately more or less diffuse.

Only in the more severe or later stages of craniocerebral injuries, however, does this sort of edema occur, the so-called "fixed edema." The usual type of cerebral edema is probably represented almost entirely by disturbances of the secretion and absorption of cerebrospinal fluid.

The cerebrospinal fluid is not static, but like the blood and lymph undergoes a process of flux. The migration of cerebrospinal fluid has been called by Cushing[4] "the third circulation," but its motion is dependent upon factors somewhat different from those which govern the circulation of the first and second circulations, i.e., the circulation of blood and lymph. The motion is secondary to a process of secretion and reabsorption. Inasmuch as the place of reabsorption is distant from the area of secretion a slow migration results.

The cerebrospinal fluid is a modified blood serum secreted, most authorities agree, through the cuboidal cells of the choroid plexus in the lateral ventricles. From these cavities the fluid passes by way of the foramen of Munro into the third ventricle and through the Aqueduct of Sylvius into the fourth ventricle. From the fourth ventricle it escapes by way of the foramina of Luschka and Magendie into the subarachnoid space which surrounds not only the brain but the spinal cord and communicates with the cisternae of the skull and the sulci of the cortex.

The mode of reabsorption of the fluid is still a matter of debate, two theories being fairly widely held; (a) that of Weed,[5] which maintains that it escapes by a process of filtration through the mesodermal cell membrane of the arachnoid villi into the venous sinuses of the skull, and (b) that of Dandy and Blackfan,[6] which maintains that it is removed by the stomata in the walls of the supracortical veins of the entire subarachnoid space, particularly by the stomata in those veins situated in the sulci and about the Pacchionian bodies along the longitudinal

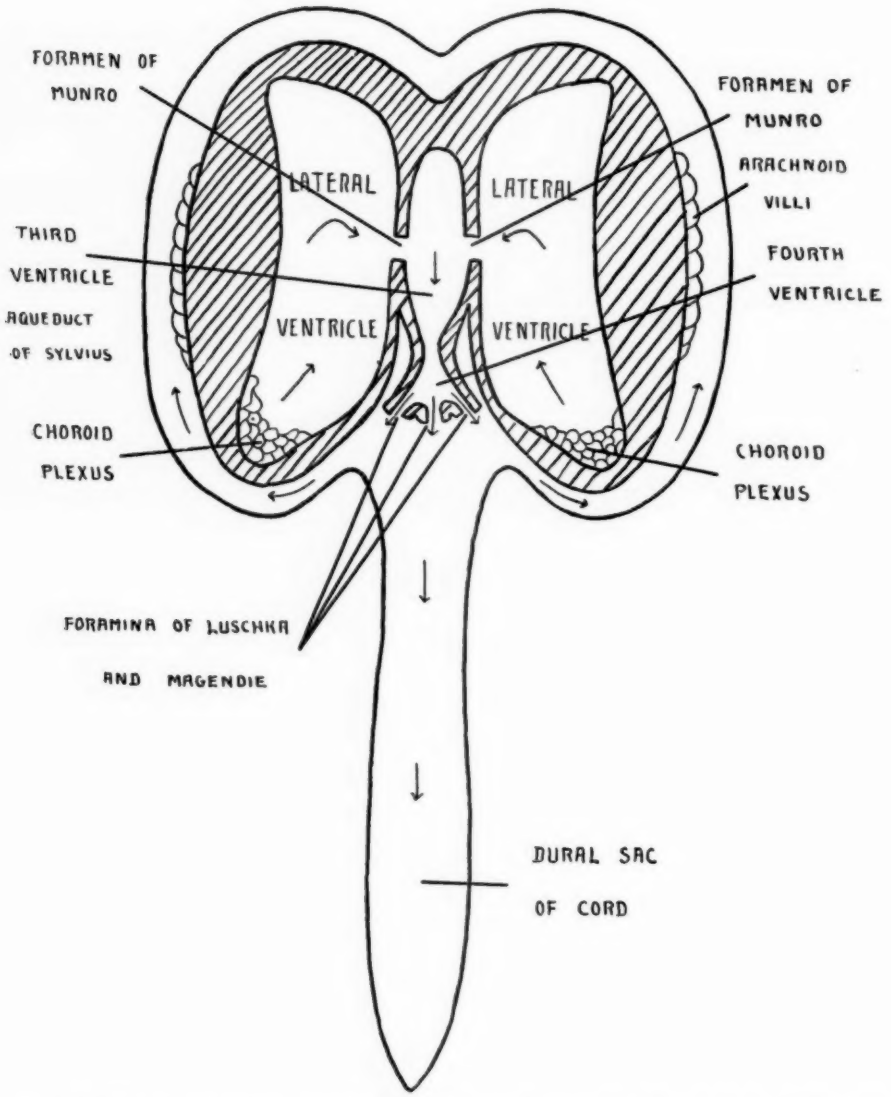


FIG. 66. Purely schematic drawing illustrating the manner of circulation of the cerebrospinal fluid, the "third circulation of Cushing." The cerebrospinal fluid is formed by a process of filtration from the blood plasma in the choroid plexuses of the lateral ventricles. It escapes by way of the foramina of Monro into the third ventricle and thence through the aqueduct of Sylvius into the fourth ventricle. From the fourth ventricle it gains access to the subarachnoid space of the brain and cord by way of the small foramina of Luschka and Magendie. It is absorbed either through the mesodermal cell membrane of the arachnoid villi or through the stomata in the walls of the supracortical veins of the subarachnoid space; absorption thus occurs on the surface of the brain and probably to some extent on the spinal cord.

sinus. Accordingly, increases in pressure within the cranium may result either from increased secretion or diminished reabsorption of the cerebrospinal fluid (Figs. 66 and 67).

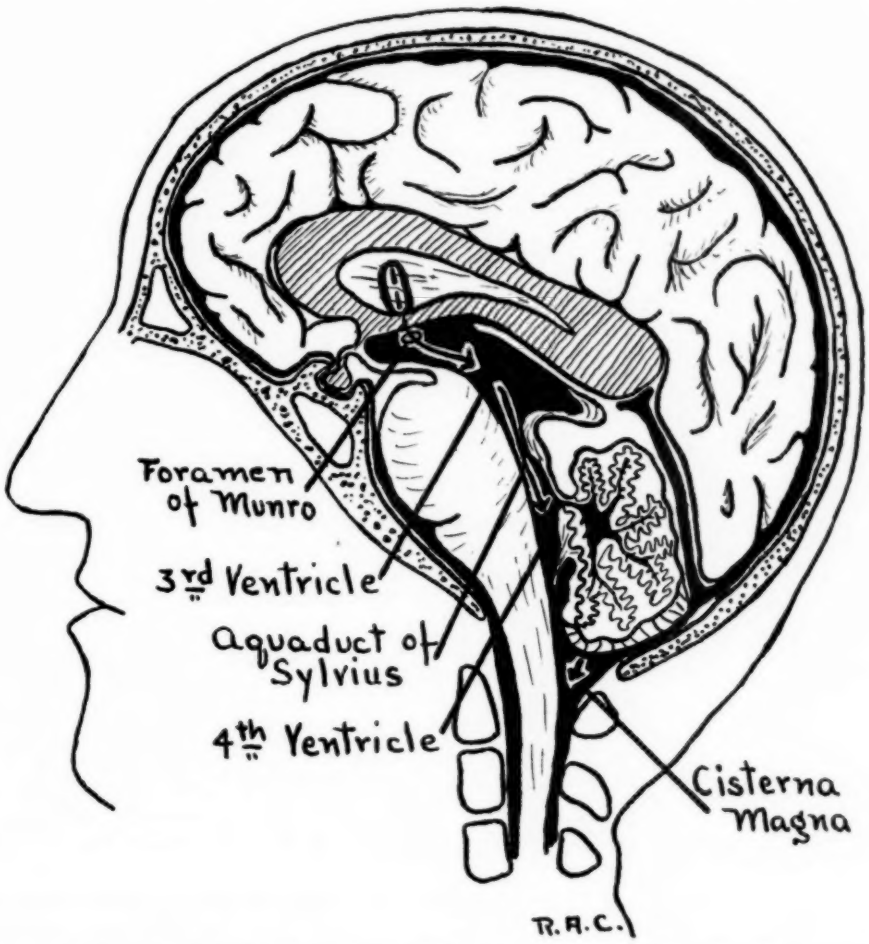


FIG. 67. Partly schematic drawing of a sagittal section of the head showing the essential anatomy of the circulation of the cerebrospinal fluid within the cranium.

The mechanism concerned in the minor degrees of head injury in which no skull fracture is sustained seems to be somewhat as follows:

The skull is slightly flattened by the impact of the injuring object, and this flattening can take place only at the expense of the cubical contents of the skull. This means that some of the contents must undergo displace-

ment into the various openings leading from the skull cavity; during this process the fine fibrillar attachments of the blood vessels are ruptured, and even the blood vessels themselves may be torn.

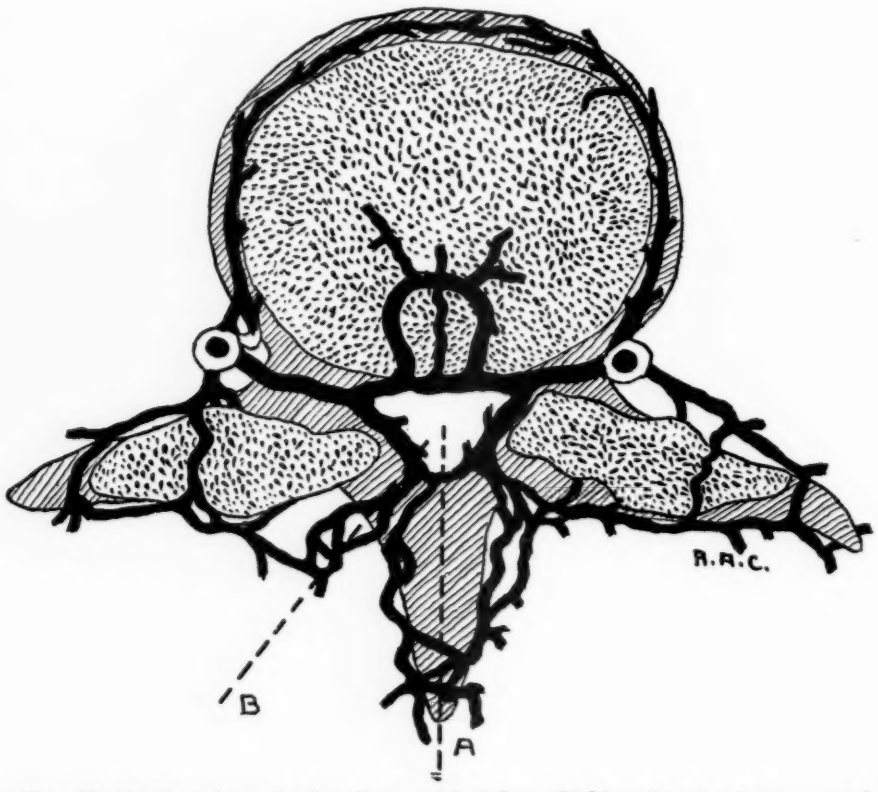


FIG. 68. Partly schematic drawing modified from Pitkin[1] showing in cross-section the anatomical arrangement of the blood supply to the cord. A spinal puncture needle introduced at one side of the midline and passing between laminae as indicated by the dotted line B is much more likely to injure sizeable blood vessels than a needle introduced properly between the spinous processes and strictly in the midline as indicated by the dotted line A.

V. The Interpretation of Symptoms on the Basis of the Pathological Physiology as Outlined

In the interpretation of symptoms arising from increased intracranial tension it is convenient to classify them according to their source, i.e., whether secondary to pressure upon the cerebral hemispheres, the midbrain, or the bulb, and also

[1] Pitkin, G. P. *J. M. Soc. New Jersey*, 24: 425, 1927.

according to their nature or severity, i.e., whether irritative or paralytic. Early symptoms of compression tend to be *irritative* and may be *localized*, as, for example, those of a subdural hematoma, a depressed fracture, or a punctate intracerebral hemorrhage, or *generalized*, as in the case of cerebral edema. Late symptoms tend to be paralytic and may also be either *localized* or *generalized*.

A. IRRITATIVE LESIONS: 1. *Cortical:* The irritative symptoms in lesions of the cerebral cortex may be either sensory or motor, or both, depending upon the region of the cortex principally involved. Hallucinations of sight, sound, and smell are not infrequent in early lesions of the occipital and anterior temporal regions, but they are not of much diagnostic value, since they are usually very transient, the patient very soon lapsing into a semiconscious condition in which the cooperation indispensable to their proper interpretation is necessarily absent.

Irritative lesions affecting the motor cortex are, on the other hand, of much greater value, because they persist even in the absence of consciousness. Muscular rigidity and spasm, fibrillary twitchings, and, in the more typical cases, true Jacksonian epilepsy, indicate involvement of the motor cortex and the pyramidal system.

2. *Midbrain:* Irritative lesions of the midbrain manifest themselves by pupillary changes, but these changes occur late, unless the primary lesion is in, or very close to, the oculomotor nuclei. Inasmuch as the nuclei of the two sides lie very close together both tend to be simultaneously involved, though not infrequently close observation will show that the symptoms on the side primarily affected develop slightly in advance of those on the opposite side. In irritation the pupil becomes contracted and reacts sluggishly.

3. *Bulbar:* The three centers primarily affected in the bulb are (1) the respiratory, (2) the cardiac, and (3) the vasomotor. Of these the respiratory is most susceptible to the effects of pressure, the vasomotor the least susceptible. Irritation of the

respiratory center produces deep, slow breathing, irritation of the cardiac center produces a slow and full pulse, and irritation of the vasomotor center increase in blood pressure and the Cheyne-Stokes type of respiration.

B. PARALYTIC LESIONS: 1. *Cortical:* Paralytic effects on the sensory cortex consist particularly of the development of unconsciousness; occasionally, however, aphasia, due to involvement of Broca's convolution of the frontal lobe may precede the development of unconsciousness, and somewhat more frequently anesthesia or analgesias of hemiplegic distribution can be elicited even in the presence of developing unconsciousness and can be evaluated by the alert observer as indicating involvement of the corresponding sensory areas of the cortex. Infrequently the development of hemianopsia is a sign which occurs before the loss of consciousness and when present is of great significance. The motor paralyses are of much more importance and consist of hemipareses and hemiplegias, exaggeration of the deep reflexes, loss of abdominal reflexes, and the Babinski and other special signs.

2. *Midbrain:* Paralytic effects on the occulo-motor nuclei produce the dilated and fixed pupil; in the early stage of paralysis the pupil, though fixed, is only slightly dilated, whereas in the later stage it is both fixed and widely dilated.

3. *Bulbar:* Paralytic effects on the respiratory center consist of irregular and shallow breathing, on the cardiac center rapid, small and easily compressible pulse, and on the vasomotor center rapid fall in blood pressure.

VI. THE SPINAL TAP AS A DIRECT MEASURE OF INCREASED INTRACRANIAL TENSION

Considering the effect of displacement of the fluid contents of the cranial cavity as the result of increased intracranial tension, it is but a step to the direct estimation of the amount of such pressure. Although blood, as well as cerebrospinal fluid, is displaced by such increased tension, the blood pressure

exhibits a very complex mechanism of readjustment to local changes of pressure. For this reason the amount of displacement of cerebrospinal fluid is taken as a guide, and this is estimated by determining the amount of rise of pressure in the communicating subdural space of the cord. Direct measurement can be accomplished by introducing a needle or cannula into the subdural space in the lumbar region and reading the pressure as indicated on an attached manometer.

The technique of lumbar puncture for purposes of estimating cerebrospinal fluid pressure is a subject which has not been sufficiently stressed in the past. For obvious reasons, in order that successive estimations be comparable either with respect to the same patient or succeeding patients, a technique must be adopted which can be standardized. Puncture in the sitting position is not feasible in a fairly large percentage of cases, and in any event the impossibility of allowing for the factor of hydrostatic pressure in such a position is enough to condemn the procedure; it is irrational to assume to measure the pressure at the top of a column of fluid by measuring the pressure at the bottom. The only standard position which all patients can assume and which eliminates hydrostatic variations is a recumbent one with the level of the ventricles the same as that of the point of introduction of the needle. With this limitation the position of the patient is a matter of relative indifference except that at least during the process of introduction of the needle he is usually required to flex the back by bringing the knees and chin as near together as possible, thus arching the back and widening the distance between the spinous processes. Whether he be required to lie on one side or the other is largely a matter of convenience.

Skin preparation is made in the lumbar region as if for a major surgical procedure, a relatively large area being sterilized. Scrupulous care is, of course, taken in the handling of sterile needles and syringes used in performing this little operation to avoid infection of the contents of the spinal canal, a circumstance which is, indeed, a calamity. An area is

selected for puncture in the midline just below a line connecting the crests of the ilia and just below the spinous process of the vertebra next above this line. This usually brings the site of puncture between the second and third lumbar vertebrae. The skin is barely pierced with a fine hypodermic needle which is directed inward at an acute angle with the skin surface, and a considerable wheal in, and not under, the skin is raised with 1.0 per cent novocaine solution. Although the subcutaneous tissues are relatively insensitive to pain, and for this reason need not necessarily be infiltrated, the patient may be saved some discomfort if the deeper layers are treated, especially the ligamentum subflavum. For this purpose the needle is now redirected so that it points directly inward in a direction perpendicular to the skin surface, and about 5 c.c. of novocaine solution are thoroughly infiltrated into the tissues as far as the hypodermic needle will reach.

The lumbar puncture proper now proceeds with the introduction of a relatively fine lumbar puncture needle through the skin wheal and in the same direction as the last insertion of the hypodermic needle, i.e., strictly in the midline and strictly perpendicular to the skin (Fig. 68). The size and shape as, well as the mode of introduction of the lumbar puncture needle, are of real importance in the avoidance of complications. Pitkin, who has devised the method of "controllable spinal anesthesia,"[7] has recently illustrated a type of needle suitable for the purpose which cuts a small trap door in the dura; this trap door closes as soon as the needle has been withdrawn after the puncture, a technique which theoretically at least seems to be capable of preventing continued seepage of cerebrospinal fluid from the puncture wound into the surrounding tissues and thus tends to minimize postpuncture headaches (Fig. 69). Punctures should be made in the midline in order to avoid miscalculations due to angular variations and also to prevent injury to veins; the bevel of the lumbar puncture needle should be short in order to insure the localization of the terminal lumen of the needle entirely within the spinal canal. The depth at

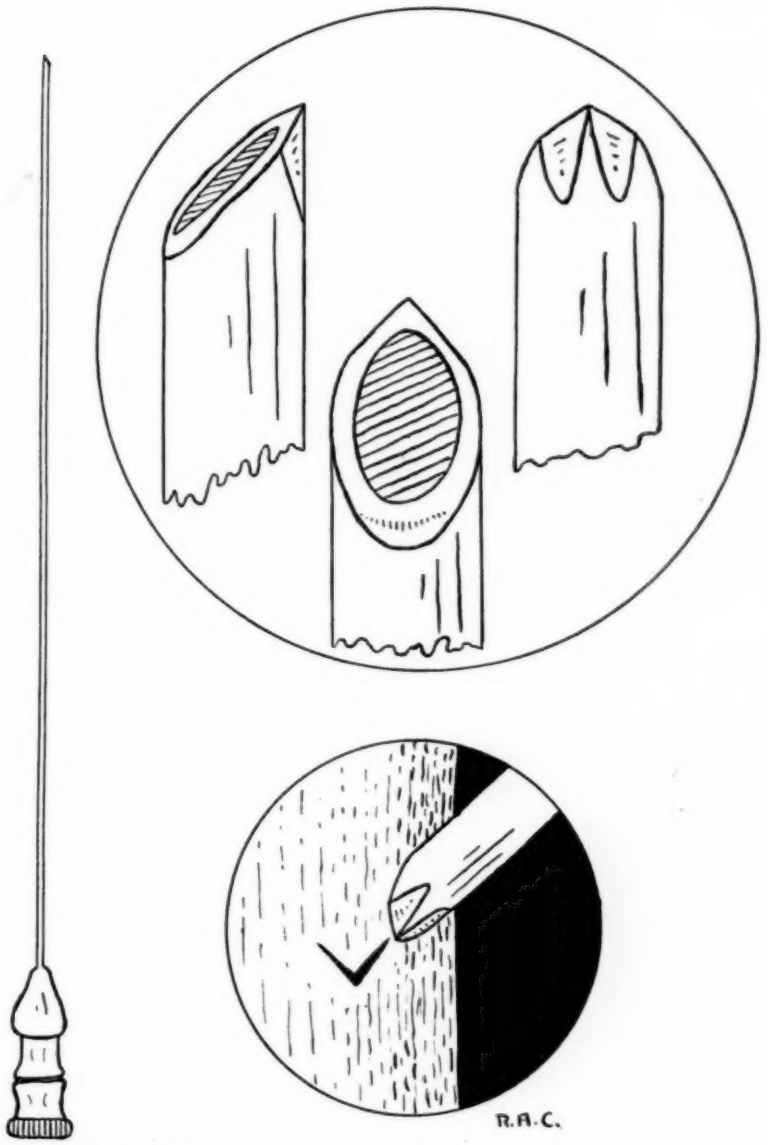
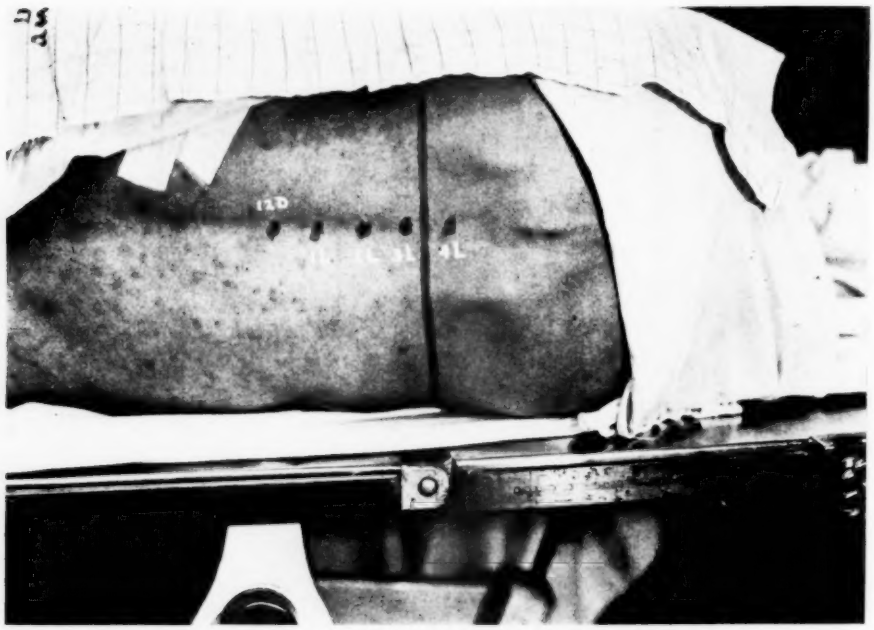


FIG. 69. Mechanical features of a specially ground spinal puncture needle (after Pitkin). On left is shown general proportions of needle; it is made of flexible steel and should be sufficiently ductile to allow bending into complete bow or semicircle. Upper insert shows magnified view of point of needle; note that the point has short primary bevel and that the shape of the point has been modified by three accessory bevels, two of these making a "v" shape at the cutting edge, and the third serving to round the edge at base of primary bevel. Lower insert shows shape of trap-door which is cut by such a needle in the dura mater; the "v" shaped flap tends to close and prevent continued seepage of cerebrospinal fluid after withdrawal of needle.

which the dura is reached varies, and it is frequently necessary to redirect the needle slightly one or more times before the canal is finally reached. A distinct feeling of resistance followed



FIGS. 70, 71, and 72. Technique of diagnostic and therapeutic lumbar puncture.

FIG. 70. Patient is lying flat on left side; knees are strongly flexed on abdomen and head and shoulders on torso. Note prominence of sacrum and rhomboid of Michaelis just to right of center of photograph; patient presents secondary rash of lues. Line has been drawn with mercurochrome between two crests of ilia, and spinous processes in lumbar region have been indicated by spots of mercurochrome; line passes ordinarily between third and fourth lumbar spines.

by a sensation of marked decrease of resistance to the progress of the needle usually indicates that the dural sac has been pierced. On withdrawal of the obturator from the needle the exudation of clear or blood-stained cerebrospinal fluid, as the case may be, indicates definitely that the puncture has been successful, and a mercury manometer is immediately attached to the needle, keeping the meniscus of the mercury always on the same level as the spinal canal in order to avoid hydrostatic effects (Figs. 70, 71 and 72).

Mere guesses as to increased or decreased pressure in the absence of accurate manometric readings when performing lumbar puncture are of no value, since the only basis on which

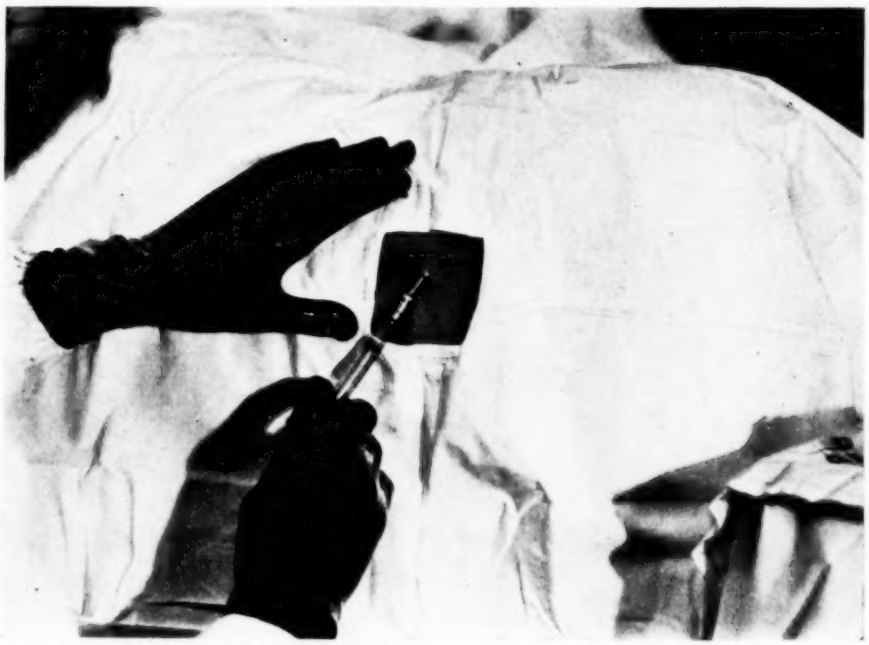


FIG. 71. Point has been selected strictly in midline and just below spinous process of third lumbar vertebra; wheal is being raised in skin by means of a hypodermic syringe, a fine needle and a 1 per cent novocaine solution; note that the patient is thoroughly draped.

guesses of this kind can be made is the rate of flow of fluid from the needle and this depends on other factors besides the factor of pressure, mainly, of course, upon the size of the lumen of the needle.

Readings on the manometer should never be taken as final for several minutes after puncture, or until several observations at intervals indicate that the pressure has become stationary. Sometimes this does not occur for a matter of five minutes, and it is not infrequently necessary to have the patient cough or to have an assistant exert pressure on the external jugular veins to make sure that there is a free flow of fluid into the manometer and that the true pressure is, therefore, being recorded.

The phenomenon of increase in cerebrospinal fluid pressure when the jugular veins are compressed is known as the "Queckenstedt Sign." It depends upon the following cycle of

FIG. 72. Spinal or lumbar puncture needle has been introduced, spinal fluid has been obtained, and a spinal manometer has been attached to lumbar puncture needle. Note manometer is being held on level with needle in order to avoid hydrostatic effects caused by weight of column of cerebrospinal fluid alone.

events, (1) the production of venous stasis in the skull, (2) increase in intracranial pressure due to this stasis, (3) and displacement of cerebrospinal fluid from the skull to the dural sac of the cord; accordingly, the test is of value in demonstrating that there is no blockage in the subarachnoid space between the point of puncture and the skull.

VII. THE TREATMENT PROPER

A. SHOCK: If patients with head injuries are admitted in shock the treatment for shock is invoked at once, the local injury being disregarded except in such cases as require the

control of hemorrhage, the precaution of covering open wounds with sterile dressings being all that is required. The fundamental treatment for shock consists in:

(a) Lowering the head (or raising the feet) to combat cerebral anemia by the action of gravity on the blood stream,

(b) Application of heat in the form of hot water bottles to the body, especially to the extremities, to combat subnormal temperature,

(c) Administration of hot drinks if the patient is in a condition to take them,

(d) Administration in the severer cases of a proctoclysis of 1000 c.c. of warm tap water (110°F) or an intravenous infusion of 500 c.c. of 5 per cent dextrose solution or physiological saline solution (110°F).

(e) Transfusion of 500 c.c. of blood in the severest cases, especially those secondary to blood loss, and

(f) Perhaps most important of all, the hypodermic administration of morphine in relatively large doses, usually half a grain at a time.

The administration of morphine should be guarded, however, lest the effect of the drug be confused with beginning coma, and in those cases in which such confusion cannot be eliminated by blood pressure readings, spinal taps, or otherwise, morphine should, of course, be withheld. Usually in the case of head injury, however, the prognosis and treatment depend not so much upon the development of symptoms as of signs, and even though certain symptoms are masked by the morphine, the signs are usually amply sufficient.

A certain number of patients never react from shock. Such patients do not regain consciousness, present a low blood pressure and rapid pulse, and pass rapidly from a condition of subnormal temperature to one of hyperpyrexia just before death. At autopsy widespread contusions of the brain substance, extensive lacerations, and free hemorrhage are characteristically found.

B. Early Treatment after Recovery from Shock: After recovery from shock the head of the patient is raised to combat the development of edema; frequently the patient may be set virtually bolt upright in bed. Of how much value the application of an ice-cap to the head may be at this stage in uncomplicated cases there is some question; such a procedure is usually grateful to the patient but probably is otherwise of relatively little value.

Two considerations now present themselves, the one having to do with the local treatment of any scalp wounds which may be present, the other with the special study of the case, particularly the skiagraphic study.

Because of the rich blood supply to the scalp, wounds in this region tend to heal kindly, but wide shaving of the scalp around lacerated wounds, debridement of wound edges, and thorough probing of wounds suspected of concealing depressed or comminuted fractures or foreign bodies should not be omitted. Primary suturing without drainage can usually be performed, especially in cases of brain injuries.

In cases in which there is hemorrhage or leakage of cerebrospinal fluid from the ears, nose, or mouth, probing and douching of these cavities should be avoided; the indications are simply to cleanse the more accessible parts and preserve the cleanliness in as simple a manner as possible.

The taking of from two to half a dozen skiagraphic plates of the skull from different angles and in different positions is of considerable importance, both from the point of view of establishing the diagnosis of such fractures as may require immediate surgical treatment and also for purposes of future reference in connection with legal medicine, workmen's compensation, permanent hospital records, and the development of complications in general.

C. Conditions Demanding Primary Radical Surgery: Three conditions demand formal operation as soon as diagnosed after reaction from shock: (1) the presence of foreign bodies within the cranial cavity; (2) the development of middle

meningeal hemorrhage; and (3) the presence of depressed fractures of the vault.

The presence of foreign bodies within the cranial cavity is not well tolerated by the organism. Local infectious processes, i.e., abscesses, are very apt to occur in and about foreign bodies, such as spicules of bone or blood clot which are allowed to remain for any considerable length of time in relation with the leptomeninges. The infectious organisms gain entrance to such a *locus minoris resistentiae* either by direct extension, in connection with compound fractures, or by way of the normal openings of the skull and especially by the blood stream. Traumatic insults that are handled easily by the frontal lobes and which result in no permanent damage may produce a rapidly fatal infection when affecting the basal structures.

Probably the best known complication of head injuries is middle meningeal hemorrhage which is recognized by the development of the typical "meningeal syndrome" as previously described. The treatment of this complication is, of course, immediate surgical intervention for the purpose of evacuating blood clot and tying the ruptured blood vessel. The presence of a depressed fracture constitutes an indication for an operation aimed at the elevation of the depressed fragment.

A sign of possible diagnostic value in localizing the side involved in obscure craniocerebral lesions is the presence of a decrease of temperature in the axilla of the affected side; this may amount to 0.5 or 1.0°.

D. The Non-surgical Treatment of Increased Intracranial Pressure: 1. *The Use of Hypertonic Solutions, Dehydration Therapy:* To Weed and McKibben[8,9] belongs credit for having first demonstrated that the volume of the brain substance can be *decreased* by the intravenous injection of *hypertonic* salt solutions or *increased* by the injection of *hypotonic* solutions and that corresponding changes occur in the pressure of the cerebrospinal fluid as measured by manometric readings taken by means of spinal puncture. These

authors used distilled water as a hypotonic agent and concentrated sodium chloride, sodium bicarbonate, sodium sulphate and dextrose as hypertonic solutions. They found a marked and sustained increase in the cerebrospinal fluid pressure after the injection of distilled water, whereas, after the injection of the hypertonic solutions there was a transitory initial increase in the pressure of the cerebrospinal fluid followed by an immediate and pronounced decrease. These changes were found independently of the amount of the solution used and were interpreted as fundamentally due to osmotic effects.

The work of these original investigators has been repeated many times and by many different authors, especially in connection with the treatment of clinical cases and accordingly may be said to have been amply substantiated.

Foley and Putnam,[10] in the year following the original reports of the previously named authors, found that the administration of sodium chloride *by mouth* accomplished the same result, and they added the hypothesis based on indirect evidence that both the intravenous and *per os* administration of hypertonic solutions not only decreases the brain bulk but also changes the ratio between the absorption and excretion of cerebrospinal fluid.

Ebaugh and Stevenson[11] and Fay[12] have shown that in addition to the initial decrease in cerebrospinal fluid pressure there is a secondary increase or "wave of edema" which occurs following the ingestion of sodium chloride by mouth, which they explain as due to a storage of sodium chloride in the brain tissue, as they did not encounter the phenomenon following the use of dextrose solution. These authors also found that the intravenous injection of 200 c.c. of a 30 per cent dextrose solution is not accompanied by the appearance of glycosuria.

Kinsman, Spurling, and Jelsma[13] have been able to demonstrate that the decrease in cerebrospinal fluid pressure which ensues upon the injection of hypertonic solutions varies in

degree directly with the concentration and amount of the hypertonic solution injected and that this decrease is accompanied by a pronounced and immediate decrease in the specific gravity of the blood serum.

Apparently, as a result of these investigations and others not specifically mentioned one is in a position to formulate the principles (1) that the injection of hypertonic solutions into the blood stream or the administration of such solutions by mouth produces an increase in the molar concentration of the blood in terms of the substances thus supplied which, in turn, results in the withdrawal of fluid from the tissues; (2) that among the tissues thus affected is the brain substance, the shrinkage of which causes concomitantly a reduction in the cerebrospinal fluid pressure; and (3) that although storage of sodium chloride in the brain tissue occurs when this substance is used as a dehydrating agent and a secondary edema follows as a result of this storage, a similar effect does not occur when dextrose solution is used. Whether the dehydrating action of hypertonic solutions is the only mechanism involved in reducing cerebrospinal fluid pressure, or whether in certain cases there is a disturbance of the secretion and reabsorption of the cerebrospinal fluid itself remains somewhat in doubt.

In the administration of hypertonic solutions by appropriate channels, is seen, therefore, an effective means of decreasing elevated intracranial tension, and the method is clinically of so much value that its invocation is urgently indicated both prophylactically and curatively. In the very mild cases of increased tension or in cases in which such a condition, though not actually present, is anticipated, a satisfactory degree of dehydration may be accomplished by the introduction of hypertonic salt solution into the bowel. Sodium chloride solution is not particularly suitable for this purpose because (1) it is very nauseating when given *per os* (2) it is absorbed from the intestinal lumen against osmotic tension (Heidenhain),[14] and (3) it tends toward the production of secondary edema as previously mentioned.

Magnesium sulphate is a much more desirable substance for enteric administration and to be effective when so used should be administered in comparatively large doses, from 2 to 3 oz. of a saturated solution by mouth or stomach tube or 6 or more ounces of a 50 per cent solution per rectum in the form of a retention enema every six hours.

In any attempt at the production of dehydration the rigid restriction of fluid intake is, of course, no less important than that of fluid extraction, since the amount of blood and tissue fluid which remains represents a balance resulting from the interplay between the two processes. This applies naturally both to the dehydration just described and also to the method of intravenous medication previously mentioned and now to be expanded. The latter method is particularly applicable to the case of moderately increased tension already developed and will be found efficacious in the treatment of many or most of such cases.

In the production of dehydration by intravenous sodium chloride therapy relatively concentrated solutions are used (25 to 30 per cent), and such solutions are not without danger, unless given very slowly (not more than 1 c.c. per minute); furthermore, secondary edema is particularly prone to occur subsequently, so that unless normal conditions can be satisfactorily restored in a sufficiently short time the last condition of the patient may actually be worse than the first. Magnesium sulphate is not, of course, suitable for intravenous injection for the purpose under discussion because of its narcotizing and toxic properties when thus employed.

Dextrose solution in concentrated form (50 per cent or more) has been found to have a very satisfactory dehydrating effect and is not open to either of these criticisms. It is, therefore, usually preferred, though relatively larger quantities must be employed (50 c.c., 100 c.c., or even more).

For hypertonic solutions of dextrose the following advantages over similar solutions of inorganic salts, particularly sodium chloride, have been claimed:

(1) They can be given relatively rapidly without danger.

(2) They do not favor the development of reactionary edema subsequently.

(3) They are more prolonged in their effects.

(4) They serve to provide a source of food energy as a secondary action.

2. *The Therapeutic Spinal Tap:* Dehydration, even by the relatively heroic intravenous methods just mentioned, does not avail in all cases. In combating cases of severer degree, or those in which the previously mentioned methods have failed, one other resort is open to the surgeon before resorting to a decompression operation, viz., relief of pressure by withdrawing cerebrospinal fluid by means of spinal puncture. The therapeutic removal of cerebrospinal fluid is a procedure of the utmost value in the more severe types of increased tension which progress unfavorably in spite of attempts at dehydration.

Jackson,[15] in 1922, advocated the treatment of all cases of head injury, save those of arterial hemorrhage and compound or depressed fracture of the skull, by means of therapeutic lumbar puncture, and was able to report a mortality in a series of 46 cases of less than 25 per cent. Munro[16] reported a series of 2908 cases of head injuries collected from 59 different clinics and treated by various other methods either combined with or exclusive of such puncture with an average mortality of 37.8 per cent, and also reported a comparative series totaling 316 cases, 217 cases of which were treated by various unselected methods with an average mortality of 22 per cent, whereas, 99 cases treated by lumbar puncture alone showed a mortality of 14 per cent.

The therapeutic withdrawal of cerebrospinal fluid is not, however, without danger, since it is possible to remove such an amount as to do away with the normal support to the brain stem, thus allowing the medulla to "jam" in the foramen magnum and produce a fatal compression of the vital centers. In order to avoid such a calamity it is never permissible to

withdraw fluid except under the guidance of carefully determined manometric readings.

When this precaution is taken, however, the procedure is eminently safe, and it is probably true that the dangers of sudden death from spinal tap have been somewhat exaggerated; deaths from this source seem to have been associated in most, if not all, cases with chronic increases of intracranial tension due to slow-growing tumors, and especially associated with such tumors as occur below the tentorium cerebelli. Shoenbeck[17] collected 71 cases of such sudden death in which 67 of the cases showed tumor or other chronic disease of the brain, and Gumprecht[18] collected 17 similar cases all showing the presence of intracranial tumors. Certainly many thousands of consecutive therapeutic lumbar punctures with drainage have been performed without proved deaths from this source.

The range of pressure recorded by lumbar puncture in head injuries is considerable and varies all the way from 100 mm. or more to normal (7 to 10 mm.); usually, of course, the higher readings are found in the more severe cases and those which are likely to result fatally.

In connection with the range of cerebrospinal pressures which may be encountered on spinal tap, the tabulations of McClure and Crawford[3] and of Jackson[15] may be cited.

	10–20 Mm. Per Cent	20–30 Mm. Per Cent	Over 30 Mm. Per Cent
McClure and Crawford	44.0	34.0	14.0
Jackson	39.8	10.8	2.0

These authors have enunciated the principle that in the performance of therapeutic lumbar puncture for increased intracranial tension *enough fluid should be withdrawn to reduce the excess of pressure over the normal pressure by one-half;* in other words, if the initial reading on the spinal manometer were 30, enough fluid would be withdrawn to reduce the pressure to 20 or 19 depending upon whether the normal pressure be taken as 8 or 10, and if the initial reading were 20, enough fluid would be withdrawn to reduce it to 15 or 14.

This principle is sound, and is generally adopted, except in cases in which more than 15 to 20 c.c. of fluid would need to be withdrawn. Although 30 or 40 c.c. (even 75 to 100 c.c.) or more have been thus withdrawn with apparent safety, it is better when possible to limit the amount to the lesser quantities and perform the operation more frequently when indicated. In this connection and with regard to the time interval between spinal taps the presence of blood in the cerebrospinal fluid withdrawn or the presence of 16 or more mm. of mercury pressure should be sufficient indication for the performance of a second tap in four hours.

Following the teaching of Cushing[4] it is customary to regard the presence of blood or a xanthoproteic reaction in the cerebrospinal fluid as an especial indication for the performance of repeated spinal punctures. It is believed that the debris resulting from blood disintegration interferes with the normal reabsorption of cerebrospinal fluid by the Pacchionian bodies. The removal of blood contaminated fluid by spinal puncture tends to prevent "clogging" of the Pacchionian bodies and thereby to obviate pressure increases secondary to diminished absorption.

Besley[19] has sounded a note of warning in connection with repeated therapeutic lumbar punctures in his observation that frequent performance of this operation may lead to stimulation of the secretory mechanism of the cerebrospinal fluid and that thus a vicious circle may become established. However, it is believed that such a development is of relatively infrequent occurrence clinically and that even though it may occur occasionally the usual effect is to break an already established vicious circle.

3. *Indications for a Decompression Operation:* Failing in the control of an unfavorable progression of symptoms and signs by all the methods previously mentioned only one resort remains, the institution of radical surgery consisting of removal of part of the cranial vault and decompression by incision of the dura, a procedure the technique of which does not fall

within the province of the present discussion. It may be mentioned in passing that the burden of clinical evidence seems to indicate that such surgery is usually of value only within the

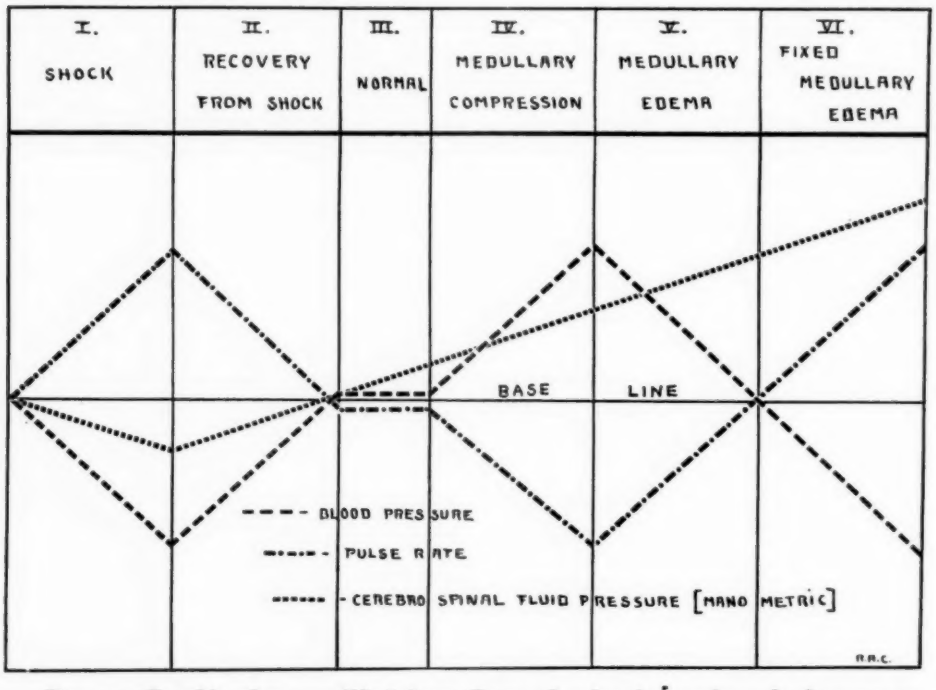


FIG. 73. Graphic chart modified from Gage, showing in craniocerebral traumata typical progression of blood-pressure, pulse rate, and cerebrospinal fluid pressure values according to six stages. Time interval occupied by various stages is variable, one patient passing rapidly from one to another, and another patient much more slowly. Any given patient may present typical progression or may show only a part of it.

first forty-eight hours following craniocerebral injuries and such patients as survive this period of time seem usually either to recover under less radical therapy or to succumb in spite of radical surgery.

E. RÉSUMÉ: As may be gathered from what has previously been said, whereas neurological examinations and a consideration of the developing symptomatology may be of considerable value in the estimation of the severity of head injuries and may serve as a guide to the manner in which a

given case may be progressing, a comprehensive understanding of such cases involves familiarity with the pathological physiology concerned and the estimation of certain variables capable of rather exact determination, pulse rate, blood-pressure, and most important of all, spinal fluid pressure.

The manner of development of these variables in a case exhibiting a typical progression of clinical manifestations has been illustrated by Gage[20] in the form of a graph which is reasonably accurate, and which serves as an admirable means of readily visualizing such a typical progression. This chart, with very minor modifications, is herewith reproduced (Fig. 73).

It will be noted that the clinical course of head injuries is divided into six stages: (1) shock, (2) recovery from shock, (3) normalcy, (4) medullary compression, (5) medullary edema, and (6) fixed medullary edema. In the columns headed by the names of these various stages are plotted the characteristic variations of (a) the pulse rate, (b) the blood-pressure, and (c) the cerebrospinal fluid pressure. In the early stages of head injury the recording of pulse rate, respiration rate, temperature, and blood-pressure every fifteen minutes for from four to six hours is of the utmost importance in determining the progress of the patient's condition. Isolated or random estimations of these variables are of but relatively little value, since normal variations are considerable; but when systematically and frequently recorded, a chart of these records gives a very accurate indication of the effectiveness of the treatment instituted and puts the medical attendant in a position to treat the case intelligently and check the development and progression of the complications which may arise. The interval between estimations after four to six hours may be gradually lengthened in cases undergoing a favorable progression of symptoms and signs.

The first stage, or stage of shock, may or may not be present when the patient is first seen; in fact, it may not

constitute a part of the clinical picture at all; it is characterized by increase of pulse rate and decrease of blood-pressure. If present, and if successfully treated by the usual methods, the stage of shock rather rapidly gives way to the stage of recovery from shock, during which stage there is a return of both pulse rate and blood-pressure to normal limits. During the stage of shock the pressure of the cerebrospinal fluid probably decreases in accordance with the depression of the blood-pressure, but inasmuch as spinal punctures are not indicated during the period of shock the exact behavior of the cerebrospinal fluid pressure during this period is of little moment.

The important point is that at the end of the stage of recovery the presence of an increased cerebrospinal fluid pressure indicates that the case is actually progressing unfavorably, whereas the presence of a normal pressure is by no means a guarantee that an unfavorable progression may not even then ensue after a variable period of complete normalcy.

The period represented by *stage three* is really the critical period in a large number of cases; it is of the utmost importance to recognize increased intracranial tension during this period, and the method *par excellence* for doing so is the method of spinal tap.

The period represented by *stage four* is the stage in which active therapy is especially indicated, dehydrative in the borderline and less severe cases, combined dehydrative and spinal decompressive in the cases of greater gravity, and combined dehydrative, spinal decompressive, and cranial decompressive in the severest cases. In cases progressing favorably blood-pressure and pulse rate return to normal, but the same series of events also occurs in those passing into the more advanced stage of medullary edema, so that the only reliable measure of the efficacy of treament in this stage consists in the measurement of the cerebrospinal fluid pressure which progressively increases in unfavorable cases, decreases in favorable ones.

The stage of medullary edema represents a distinctly serious condition in which the prognosis becomes a matter of grave concern. No method of treatment can be depended upon to curb the progression of symptoms in this stage; dehydration, repeated spinal tap, and cranial decompression may be and, of course, should be tried, but the stage of fixed medullary edema tends to ensue in spite of treatment.

The stage of fixed medullary edema is a terminal affair, and in this stage treatment of any kind whatever is of no avail.

VIII. Prognosis

The prognosis of any but the slightest degrees of head injury should be extremely guarded. Since the incidence of chronic subdural hematoma and gliosis of the cortex has come to be appreciated and a variety of vague major and minor symptoms, accompanied by chronic increases of intracranial pressure, have been described, it has been generally recognized as highly unwise to express in connection with the acute case a very definite opinion as to the eventual outcome of any type of serious head injury. The symptoms of chronic subdural hematoma, arising as they often do from six weeks to six months or more after the original injury, and the symptoms of chronically increased intracranial tension being noted, often for the first time only after several months or even years (Sharpe[1]), makes an even comparatively delayed expression of opinion a very hazardous affair. The return of the functions of injured cranial nerves may be very much delayed, and the ultimate outcome is nearly always a matter of very grave doubt.

As to the prognosis in the severe types of injury, as it concerns the life of the patient, only as a result of the very careful weighing of all the evidence can any reasonably accurate estimation be formulated. However, there are certain rather ominous symptom-complexes which may be mentioned and which, if recognized, will immediately indicate to the

medical attendant's mind that the patient's life is in immediate peril:

1. The presence and persistence of dilated or fixed pupils,
2. The early development of the Babinski toe sign,
3. The presence of a persistently bloody cerebrospinal fluid.
4. A steadily rising pulse rate and blood-pressure with increasing restlessness or deepening coma.

Other things being equal, the immediate prognosis in head injuries seems to be somewhat better for children than adults; not only is the percentage of recoveries greater but the per-percentage of complications less, according to Moorhead and Weller.[21] Children are, however, more prone to develop late sequelae, such as mental retardation, epilepsy, and spastic paralysis.

Because of the likelihood of the development of untoward complications and sequelae, victims of head injuries are required to remain in bed and under observation in hospitals for much longer periods now than formerly and are advised to report for "follow-up" examinations for a period of at least a year.

The incidence of the late complications of cranial injuries which are found after months or years and consist of persistent headache, mental depression or excitability, change of personality, convulsions, and a host of lesser complaints, has been made the subject of a statistical study especially by Sharpe[1] who attempted to trace the subsequent history of cases of traumatic head injuries discharged from four of the larger hospitals of New York City during the ten-year period from 1900 to 1910. Only 34 per cent of the patients could be traced, but of these 68 per cent complained of symptoms presumably referable to the previous head injury and 50 per cent showed actual objective evidence of increased intracranial tension as shown by manometric cerebrospinal fluid pressure readings.

Although many competent clinicans more or less discredit the etiological relationship between the craniocerebral injury and the development of psychoneurotic manifestations subse-

quently, prefering to incriminate the factor of suggestion rather than any organic lesion, it is probably best to preserve an open mind in the matter until the question has been subjected to more careful investigation. The last word has by no means been spoken at the present time.

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Chapter XXIII

THE PREOPERATIVE AND POSTOPERATIVE CARE OF PATIENTS WITH INTESTINAL OBSTRUCTION

CONTENTS OF CHAPTER XXIII

A. J. S. PAGE

I. Definition and classification of ileus 763
II. The clinical picture in ileus 767
A. Constipation 767
B. Vomiting 768
C. Pain 768
D. Temperature, pulse, and respiration 768
E. General appearance of patient 768
F. Laboratory examinations 769
III. The diagnosis of ileus 769
IV. The pathology of ileus 773
V. Metabolic disturbances 774
A. Dehydration 775
B. Alkalosis 775
C. Hypochloremia 776
D. Increase of blood non-protein nitrogen 777
VI. The treatment of ileus 778
A. Prophylactic treatment 778
B. Active treatment 779
1. Reestablishment of intestinal flow 779
a. Methods suitable in dynamic ileus 780
b. Methods suitable in adynamic ileus 781
The rationale of enterostomy 782
Drug therapy in ileus 785
Splanchnic and spinal analgesia 787
C. The treatment of the toxemia of ileus 790
1. The use of water, sodium chloride, and dextrose 791
2. The use of gastric lavage and external heat 792
3. The use of perfringens antitoxin 792
VII. Prognosis in ileus 793
References 794

Chapter XXIII

THE PREOPERATIVE AND POSTOPERATIVE CARE OF PATIENTS WITH INTESTINAL OBSTRUCTION

1. Definition and Classification of Ileus

Intestinal obstruction, or ileus, may occur either as the primary disease which sends a patient to the surgeon or as a complication in the postoperative convalescence of patients subjected to operations for other reasons, especially in those who have undergone previous laparotomies.

Postoperative ileus is due in almost all cases either directly or indirectly to peritonitis; about one-half of all cases of ileus are postoperative, and the condition of appendicitis accounts for by far the greatest number of cases of postoperative intestinal obstruction.

Cases of postoperative ileus occurring (1) within a few days of an operative procedure are usually directly dependent upon acute peritonitis either localized or generalized, and the obstruction is caused by agglutination, by plastic exudate, of one or more coils of intestine to other coils of intestine, or other intra-abdominal viscera, (2) cases which occur later than this but still within the period of convalescence are usually due to residual abscess formation, and (3) cases which occur after the period of convalescence, occasionally a number of years afterward, are due to strangulation of intestinal viscera by bands of organized adhesions.

Unfortunately for the ease with which the diagnosis of ileus may be made in cases which develop ileus during the period of convalescence from laparotomies the symptoms consist in the early stages merely of abdominal distention, pain, and nausea. Inasmuch as these three symptoms occur to a greater or less extent in most patients convalescing from laparotomies the differential diagnosis between incipient ileus and a number of other postoperative com-

plications like gastric dilatation, dehydration, disturbances of the acid-base balance, and general peritonitis, is usually exceedingly difficult. The diagnosis must be made very largely by a process of exclusion, and frequently the diagnosis can be made with reasonable certainty only after the lapse of a number of hours, and after energetic employment of gastric lavage, hypodermoclysis, and intravenous infusion, and the usual measures for the relief of distension have failed to produce a satisfactory improvement in the patient's condition.

The diagnosis of intestinal obstruction should always be considered and excluded in all cases of appendectomy in which after a period of relatively normal postoperative reaction, nausea and vomiting return, the pulse rate increases and flatus ceases to be passed. Pain may or may not be a prominent feature in cases of postoperative ileus: when severe and colicky the possibility of strangulation should always be considered.

Sir Samson Handley believes that in generalized peritonitis death is never due to the peritonitis itself but always to intestinal obstruction.[2] He believes that generalized peritonitis always starts in the pelvis and rises in the abdomen like a flood progressively causing peritoneal irritation and paralysis of the loops of intestine as it goes. Death of the patient usually ensues before the advancing process has involved the intestinal coils much above the level of the umbilicus.

Ileus may be defined as an interruption to the flow of intestinal contents through the gut together with perverted motor responses in the intestinal musculature and associated altered metabolic reactions. Ileus is classified into two varieties: (1) the mechanical or dynamic variety, and (2) the paralytic or adynamic variety.

Dynamic ileus occurs whenever there develops some mechanical barrier to the normal flow of intestinal contents through the small or large bowel. Such obstruction of intestinal contents may occur as the result (a) of pressure applied to the intestinal tract from without, as in the case of tumors, misplaced normal organs, or bands of scar tissue and adhesions;

(b) encroachments upon the lumen of the gut, arising from the wall of the intestine itself, usually as the result of tumor formation either benign or malignant; and (c) the presence of foreign bodies within the lumen of the gut, such as, fecaliths, large impacted gallstones, or other foreign bodies. Dynamic ileus may be partial or complete. In its simple form it is accompanied by no decrease in intestinal motility, in fact, the motility of the intestines proximal and even distal to the obstruction is characteristically exaggerated, and the resulting violent movements tend to overcome the barrier to the progress of intestinal contents and so reestablish normal function.

Paralytic ileus may arise under certain circumstances spontaneously and in the absence of any mechanical obstruction to the lumen of the intestines, or it may constitute the secondary or late stage of a preexisting dynamic ileus. It is a condition in which the progress of intestinal contents along the intestinal tract is arrested because of paralysis of intestinal movement; the musculature of the wall of the gut is in a relaxed, atonic, and non-motile condition and there is, therefore, no tendency to overcome a mechanical barrier to the onward progress of intestinal contents if such a barrier be present. The mere presence within the lumen of the intestine of products of digestion which fail to make satisfactory progress toward the anus constitutes a passive condition of simple constipation; ileus, on the other hand, represents the active response of the organism to intestinal stasis.

Murphy and Brooks,[3] Hartwell and Hoguet,[4] Wilkie,[5] and others have presented evidence to show that the essential factor in the production of ileus is not blockage of the lumen of the intestine, but interference with the blood supply of the intestinal wall; only when such vascular disturbance occurs do the various phenomena characteristic of ileus develop.

An attempt has been made recently by Owings, McIntosh, Stone and Weinberg[6] to explain interference with the blood supply on the basis of increases in intra-intestinal pressure. Though these particular investigators found the pressure in

various parts of the intestinal tract to be similar regardless of the site of the obstruction and therefore were unable to explain certain characteristic differences between "high" and "low" obstruction, Morton[7] states that he has been able to demonstrate that the pressure in the duodenum is six or seven times as high in duodenal obstruction as the pressure within the ileum in ileal obstruction. This he believed he could explain on the basis of the fact that more material is secreted into the lumen of the duodenum than is secreted into the ileum (or jejunum) and this he believed would account for the relative severity and suddenness of onset of symptoms in high obstruction; nowhere else in the intestinal tract is so much secretion found as comes from the ampulla of Vater in the form of biliary and pancreatic secretions.

Dragstedt, Lang, and Millet[8] have attempted to incriminate the anatomical topography of the intramural blood supply as of direct etiological significance in the development of ileus. They believe that increases of intra-intestinal pressure are responsible for circulatory embarrassment, but that the degree of embarrassment caused by a given increase in intra-intestinal pressure is dependent upon the relative length of the veins coursing through the intestinal wall; the intramural portion of the veins is longer in the upper intestinal tract and is occluded much more readily than the intramural portion of veins which occur more distally and are shorter. They present good evidence that an intra-intestinal pressure of 35 or 45 mm. of mercury is quite safe within the ileum and colon, whereas within the duodenum and the upper jejunum such a pressure immediately produces venous collapse.

Raine and Perry[9] have produced additional experimental evidence to show that the essential factor in ileus is embarrassment of the blood supply due to increased intra-intestinal pressure. They found that whereas the administration of water to rabbits in which experimental obstruction had been performed did not appear to influence the course of their condition unfavorably, the administration of liquid albolene

caused a very rapid development of serious symptoms and signs. The explanation offered is that water is absorbed rather readily from the intestinal tract, such absorption keeping the intra-intestinal pressure relatively low, whereas liquid albolene, not being absorbed at all, produces rather prompt increases in intra-intestinal pressure due to its bulk.

It is both clinically and experimentally true that obstruction high in the intestinal tract is very much more speedily followed by the symptoms and signs of disease than intestinal obstruction in the lower portion of the small intestine or in the colon. In fact, it would be essentially true to state that the time of onset and severity of the symptoms of intestinal obstruction vary, the former directly and the latter inversely, with the distance between the pylorus and the site of obstruction. Possibly the foregoing considerations account in whole or in part for this difference in reaction.

II. The Clinical Picture in Ileus

Patients with obstruction in the small intestine rarely survive for more than six or eight days unless the obstruction is overcome by suitable means, and even then such patients may survive indefinitely or succumb, largely depending upon the duration of the obstruction at the time it is relieved.

Obstruction in the duodenum or uppermost jejunum may prove very much more rapidly fatal, patients usually surviving such obstructions for only two or three days. Obstruction within the large intestine is not inconsistent with life unless prolonged for very much more considerable periods of time, occasionally for several weeks or even two or three months.

The only two invariable signs of intestinal obstruction are constipation and vomiting.

A. Constipation: Constipation does not, of course, become absolute at the time obstruction occurs; inasmuch as the intestinal canal below the point of obstruction may contain products of digestion and may continue to function normally for a variable period of time the patient may expel

several apparently normal stools; flatus may also be passed as long as the bowel below the point of obstruction still continues to function.

B. VOMITING: Vomiting is the most characteristic sign of obstruction. At first it is accompanied by retching, and the vomited material initially expelled consists of gastric and duodenal contents. Later the vomiting becomes passive and the vomited material may spill out of the mouth without apparent effort on the part of the patient. As the condition progresses and in case the obstruction is low in the intestinal tract, the contents of the jejunum, ileum, and colon are ejected in the order named, and in cases of low obstruction the vomited material may eventually become even fecal in character. Fecal vomiting, however, constitutes no essential factor in the timely diagnosis of obstruction. It is, as has been aptly remarked, a "herald of approaching death."

C. PAIN: Pain may or may not be a prominent symptom, depending upon whether the ileus is obstructive or paralytic in type. In obstructive ileus there is characteristically acute, intermittent, colicky, abdominal pain, similar to that produced by obstruction of any other hollow abdominal viscus. Adynamic or paralytic ileus is characteristically painless in its onset and course.

D. TEMPERATURE, PULSE, AND RESPIRATION: The temperature of a patient suffering from intestinal obstruction is characteristically either normal or subnormal. The pulse, normal at first, becomes progressively weaker and more rapid. Associated with the weak and rapid pulse is an abnormally low blood-pressure. Respirations become progressively more and more shallow.

E. GENERAL APPEARANCE OF PATIENT: In the early stages of ileus the general condition of the patient is comparatively good. In a typical and fully developed case the patient appears acutely ill, his eyes are bright, his cheeks are sunken, and he may present the typical "facies hippocratica." The extremities are cold and clammy, and the hands and feet present fibrillary

twitching. Beads of cold perspiration occur on the forehead; the lips and nail beds are frequently cyanotic. In the terminal stages a deep coma may supervene and this is rather rapidly followed by death. Previous to this time, however, the patient may be unusually alert mentally, and acutely aware of his surroundings. He usually tosses about in bed, may talk of trivialities, and make plans for the future. He rarely recognizes the seriousness of his condition unless specifically warned.

F. LABORATORY EXAMINATIONS: Patients with intestinal obstruction characteristically have a scanty urine, which shows traces of albumin and frequently a few casts. The non-protein nitrogen is characteristically increased from two to seven times the normal amount. Urinary chlorides are proportionately reduced. Kidney function tests, such as the Rowntree-Geraghty test, may show slightly decreased or even normal urinary output. The red blood cell count is normal or somewhat increased. There is characteristically a leucopenia. The blood chemistry findings are characteristic. The blood. chlorides are decidedly reduced. There is an increase in non-protein nitrogen which is due almost entirely to an increase in the urea content of the blood. The creatinine may also be increased. The carbon dioxide combining power is characteristically markedly increased.

III. THE DIAGNOSIS OF ILEUS

The diagnosis of intestinal obstruction is made on the basis of the history, the symptoms as previously outlined, and the laboratory findings. The physical examination of patients with intestinal obstruction, except for some inconstant finding such as the presence of an external hernia the contents of which cannot be reduced, is of relatively little consequence. A general discussion of the causes and differential diagnosis of intestinal obstruction need not be here attempted, because such a discussion more properly falls within the scope of general surgical diagnosis. Ileus as a postoperative complication, however, requires especial consideration.

FIG. 74.

FIGS. 74 and 75. Ordinary "flat plates" of patients with early ileus, showing multiple fluid levels surmounted by gas bubbles. These skiagrams were taken with the patients in the upright position, but when patients are too ill to sit upright the same effect may be obtained with the patient lying on the side or the back by directing the *x*-rays horizontally through the abdomen against a plate held vertically.

Inasmuch as one of the most fruitful causes of postoperative ileus is the presence of angulations and strictures caused by the presence of postoperative adhesions, the diagnosis of ileus

FIG. 75.

should be considered in all cases in which suspicious signs and symptoms occur in patients who have undergone either recent or remote laparotomies. Loops of intestine frequently become adherent to abdominal wounds, and the site of the abdominal

incision may sometimes give a valuable clue to the site of an obstruction. The occurrence of cramps or colicky intra-abdominal pains always indicates that peristalsis is active, and by his sensations the patient is frequently able to localize, with some accuracy, the relative location of the obstruction. It is important to realize that the small intestine is the most frequent site of mechanical obstruction and this is doubtless largely due to the length of its mesentery.

The area of maximal distention in an abdomen, the seat of obstruction, is frequently a valuable indication of the part of the small intestine involved. Obstruction low in the ileum characteristically produces abdominal distention in the lower part of the abdomen; the upper part of the abdomen is relatively flat. Distention in the lower right abdominal quadrant usually indicates that the obstruction is in the low, or terminal portion of the ileum, whereas distention occurring particularly on the left side of the abdomen characteristically indicates obstruction in the jejunum. These considerations apply only to the dynamic type of ileus, for in paralytic ileus distention is characteristically more generalized over the entire abdomen. Obstruction high in the jejunum and obstruction in the duodenum characteristically produce no distention at all.

A type of postoperative ileus seldom considered is that produced by previously existing but unrecognized intestinal carcinoma. Although the ileus produced by a carcinoma of the gut is insidious in its onset in two-thirds of the cases, the other one-third produce symptoms of acute obstruction. The possibility of the occurrence of such an obstruction during the course of postoperative convalescence should not be forgotten, especially in patients past the age of forty years. Crile[10] is authority for the statement that, apart from the development of mechanical obstruction and peritonitis, the occurrence of postoperative ileus bears a direct relationship to the amount of trauma inflicted on the viscera during the course of any operative procedure. It is well to bear this observation in mind when the diagnosis of obstruction comes in question.

In both types of obstruction a diagnostic procedure of the utmost value in cases of doubt consists in the skiagraphic examination of the abdomen of the patient in the upright position. As is well known, the administration of contrast substances to a patient with suspected ileus is a distinctly dangerous procedure because of the possibility of converting a partial obstruction into a complete one as the result of an accumulation of contrast material in the constricted portion of the intestinal lumen.

Such a procedure is unnecessary in cases of obstruction of the small intestine, as first emphasized by Schwarz,[11] in 1911, who showed that in cases of ileus the intestine contains both fluid and air, and an ordinary flat plate with the patient in the upright position is capable of showing the presence of multiple fluid levels surmounted by accumulations of gas (Figs. 74 and 75). The procedure is well known in the European clinics, but has rarely been called to the attention of surgeons in the United States, although Case[12] has been partly successful in bringing this procedure to the attention of American surgeons.

In obstruction to the colon there is usually, of course, no contraindication to the administration of a barium enema, and this may occasionally localize the point of obstruction with great accuracy. Roentgenographic examination of the abdomen by one or the other of these methods may be diagnostic within six hours of the onset of ileus and is accordingly one of the most valuable early diagnostic measures which can be employed.

The use of methods designed to stimulate intestinal motility and the elimination of a diagnosis of ileus on the basis of subsequent bowel evacuations is a distinctly dangerous procedure because the lower bowel may evacuate its contents even though an ileus be present in the proximal portion of the gut.

IV. The Pathology of Ileus

Gross or microscopical changes in the various organs of the body are remarkably absent in patients dying from ileus.

Most of the characteristic changes are found in the intestine itself; the splanchnic area shows extreme vascular engorgement; the intestine above the site of obstruction, if not ruptured, is dilated; the intestinal wall is dusky or bluish-red in color. There may be petechial hemorrhages both beneath the serosa and on the mucosa. The lumen of the intestine is characteristically filled with foul-smelling brown or reddish-brown fluid, and the bacterial content can be shown to be much increased over normal. Microscopically the intestinal wall shows remarkable capillary engorgement and occasional areas of necrosis, especially above the site of obstruction.

Fatty changes in the liver and microscopical evidences of toxic nephritis have been reported, but are not universally found. In experimental animals, Cutting[13] has found the adrenal glands increased to from three to six times their normal size, and on microscopic examination of the cortex has found multiple areas of focal necrosis with hydropic degeneration of the cells and pycnosis of the nuclear material. In the medulla the chromaffin material was found greatly reduced, and there was an infiltration of lymphoid and plasma cells.

The essential abnormalities found in ileus are considered not to be essentially anatomical, but rather physico-chemical.

v. Metabolic Disturbances

The physico-chemical changes occurring in ileus together with the symptoms dependent upon such changes have been usually designated collectively as the "toxemia" of intestinal obstruction. The experimental and speculative literature on the "toxemia" of intestinal obstruction has become remarkably prolific within recent years. A review of all of this literature would in itself constitute a rather considerable monograph and could scarcely be undertaken with profit in this connection, as many of the observations which have been made present no clear-cut therapeutic indications in the present state of their development.[12]

Four definite and significant physico-chemical disturbances have been sufficiently demonstrated. All of these changes increase progressively with the development of the ileus, and all of them tend to be more rapid and profound when the obstruction is high in the digestive tube: (A) dehydration, (B) alkalosis, (C) hypochloremia, and (D) increase in the non-protein nitrogen of the blood.

A. DEHYDRATION: Hartwell[4] has apparently shown rather clearly that the dehydration found in ileus is the direct result of vomiting. As Braun and Boruttau[15] have emphasized, the normal intestine produces within twenty-four hours an amount of secretion almost equivalent to the entire amount of blood and lymph contained within the body. The dilated and partially strangulated intestinal wall proximal to the site of obstruction is incapable of reabsorbing the secretions which are poured into the lumen, regurgitation and vomiting occur, and thus is reversed the normal process by which continual and progressive dehydration is prevented.

In the terminal stages the toxemia of ileus produces the characteristic picture of shock; the peripheral capillaries undergo extreme dilatation, and the patient develops profuse cold sweating which still further depletes the body of its water content.

B. ALKALOSIS: Hastings, Murray, and Murray[16] were apparently the first to demonstrate that the carbon dioxide combining power of the blood is unusually high in intestinal obstruction. It has been stated that the chlorine ion accounts for 50 per cent of the acid forming elements in the normal blood. The gastric mucosa secretes hydrochloric acid, which, of course, contains chlorine, and the liver and pancreas both secrete quantities of chlorine in the form of sodium and other chlorides. Whether these chloride ions are vomited and thus actually discharged from the body or whether they merely accumulate in the dilated intestine, as in the case of animals like the rabbit which cannot vomit, the effect is the same. If the loss of the acid radical, chlorine, occurs so rapidly and exten-

sively that increases in the acid carbonate radical cannot compensate for them, alkalosis results. MacCallum, Lintz, Vermilye, Legget, and Boas[17] have formulated the idea that the hyperexcitability and tremor frequently shown by patients with excessive vomiting are actually a manifestation of alkalosis, and on this basis have suggested alkalosis as the cause of gastric tetany.

c. Hypochloremia: Various explanations have been suggested to account for the hypochloremia of ileus: (1) the fasting usually incident thereto, (2) the concomitant increase in body temperature, (3) the effect of alkali therapy, (4) altered renal threshold, (5) vomiting, and (6) detoxication retention. Most of these factors can be dismissed summarily as unimportant.

In normal individuals the feeding or withholding of chlorides affects the concentration of chlorides in the blood little or not at all. Increases of body temperature do not regularly or extensively lower the renal threshold for chlorides, and in any case fever rarely occurs as a symptom of uncomplicated ileus. Hypochloremia occurs even in those cases in which alkali therapy is not adopted and therefore failure to provide alkali therapy cannot be accepted as explanatory. Haden and Orr[18] do not believe that vomiting accounts for decreases in the chloride content of the blood, because they found consistently that such changes occurred in rabbits and monkeys, both of which animals do not vomit. As suggested by Cooper,[14] however, these observations fail to take into account the considerable quantities of fluid which can accumulate in the dilated portion of the intestine proximal to an obstruction. Haden and Orr[19] have offered the explanation that chlorides are removed from the blood and become fixed within the tissues in combination with toxic bodies. They believe that these toxic bodies are neutralized by this process and become innocuous. Although this explanation has much to recommend it, the administration of chlorides in forms other than sodium chloride is apparently of no value. Furthermore,

Gatch, Trussler, and Ayers[20] found on careful examination that the tissue chlorides, as well as the blood chlorides are reduced and in similar proportions.

D. INCREASE OF BLOOD NON-PROTEIN NITROGEN: The blood non-protein nitrogen increase which is observed characteristically in intestinal obstruction is very largely due to increased urea concentration. This phenomenon was first described by Tileston and Comfort[21] in 1914. It immediately suggests impaired kidney function, but impairment of kidney function is not the cause of the condition, for the increased urea concentration can be shown to be due not to retention but actually to increased formation of urea. Haden and Orr[22] and Whipple, Cook, and Stearns[23] have shown that nitrogen excretion in the urine is not diminished in cases of ileus but is actually increased to four or five times the normal value. McQuarrie and Whipple[24] have shown that the kidney may be damaged slightly, but that the amount of damage is inconsiderable.

The fact that the kidneys in ileus are capable of secreting so much urea also serves as a very good indication that kidney damage is no essential part of the picture. The finding of excessive amounts of urea in the urine sufficiently disposes of the contention that increases in blood urea are entirely secondary to dehydration and concentration of the blood. Since urea can be derived in no other way than from destruction of proteins or amino-acids, one is forced to conclude that in intestinal obstruction there occurs an exaggerated catabolism of the protein molecule somewhere in the body. Whipple and his co-workers[25] explain this on the basis of the action of some toxic body on the tissues, and they believe that the administration of quantities of fluid dilutes the offending toxin and thus produces diminution of protein cleavage. Hartmann and Smyth[26] suggest the rather nebulous hypothesis that urea appears in the blood to compensate for the loss of chlorides. They imply that it has some action either in stabilizing the acid-base balance or in maintaining proper osmotic relations.

Bacon, Anslow, and Eppler[27] believe that in dehydration the water of hydration is removed from the protein molecule to stabilize the water metabolism; the dehydrated protein molecules then disintegrate with the resultant production of excessive quantities of urea. However, in those cases in which they were able to produce comparable phenomena experimentally by methods other than the production of ileus, there was a marked hyperpyrexia, which, of course, does not occur in obstruction. It must probably be admitted that the explanation of this part of the picture of ileus is as yet obscure.

VI. The Treatment of Ileus

A. Prophylactic Treatment: The prophylaxis of ordinary primary ileus is discussed in all textbooks in general surgery and need not be here repeated. The prophylaxis of postoperative ileus, however, may profitably receive brief consideration. Ileus was formerly a much more formidable postoperative complication than it is at present. Many cases developed on the basis of a preexisting localized or generalized peritonitis, and a considerable number of others apparently were directly due to faulty technical manipulations within the abdominal cavity. As the science and art of surgery have been improved the occurrence of postoperative ileus has proportionately decreased. It, therefore, seems obvious that the occurrence of postoperative ileus is favored (1) by delay in the recognition and treatment of disease processes which characteristically eventuate in the development of peritonitis and (2) by inexpertness in technical surgical procedures. Considerations of especial importance in operative technique include: avoidance of undue manipulation of the contents of the abdomen, care in the reperitonealization of denuded areas in an effort to prevent the development of extensive adhesions, careful suturing of the abdominal wall to prevent postoperative herniation and wound rupture, and the more obvious avoidance of the production of any condition which might produce or predispose to

angulation, kinking, or other factors tending to produce mechanical occlusion of the intestinal canal.

Certain patients seem to show a particular tendency to excess connective tissue formation within the abdomen after laparotomy; the plastic exudate which always forms on loops of intestine subjected to trauma, and which in most patients is subsequently liquefied and absorbed, in patients showing this "keloid tendency" undergoes organization and contraction with age. Such patients are especially subject to "postoperative adhesions," and although such a keloid tendency cannot, of course, be recognized at the time of primary laparotomy, it should be considered in connection with secondary laparotomies for the relief of adhesions, and in such cases particular care should be used in order that the last condition of the patient may not be actually worse than the first. Attempts to combat the re-formation of adhesions by the introduction of various substances into the abdominal cavity at the time of operation have usually been relatively unsuccessful. The use of vegetable or animal "digestants" seems to hold some promise of success, papain and possibly trypsin, and also the use of amniotic fluid or some extract from such fluid.

B. ACTIVE TREATMENT: The curative treatment of ileus as at present understood consists of two parts: (1) The reestablishment of normal intestinal flow through the gut, and (2) the treatment of associated metabolic disturbances, i.e., the relief of toxemia. Although these two aspects of the treatment must be discussed separately they are actually both equally important and must be considered in clinical cases not seriatim but in close conjunction.

1. *Reestablishment of Intestinal Flow:* Although no treatment of intestinal obstruction can possibly avail which does not include restitution of the normal flow of intestinal contents toward the anus, the mere reestablishment of the flow may or may not insure the patient's recovery, depending upon the duration and degree of associated toxemia. Clinically, the evacuation of an obstructed loop or segment of intestine,

however accomplished, may either establish the process of recovery or may very soon precipitate the death of the patient. Characteristically, patients who have been relieved of ileus present within a very short time of the restoration of intestinal function an improved appearance which usually marks the beginning of a progressive change for the better, but in certain cases this phase may speedily be followed by a reversal of the clinical picture and the exitus of the patient. Apparently it is possible to precipitate a patient's demise by the sudden restitution of normal activity to an intestine which has been the seat of obstruction for a prolonged period of time. In the treatment of intestinal obstruction, accordingly, promptness in diagnosis and treatment is of the essence of success.

a. Methods Suitable in Dynamic Ileus: Dynamic ileus is probably never fatal unless it progresses to the paralytic or adynamic stage, and the treatment of this condition is therefore always surgical. The particular procedure to be performed and the technique of performing it depend to a considerable extent upon the variety of obstruction with which one has to deal. In the performance of any operation on patients with intestinal obstruction, however, a preliminary estimation of the severity of the associated toxemia should be made and if toxemic manifestations are definite, as they will almost invariably be in all cases except those suffering from colonic obstruction of relatively short duration, the rule should be to do only as much surgery as is absolutely necessary and no more. Frequently this will involve little more than the performance of enterostomy; sometimes it may involve the exteriorization of an intestinal tumor which under more favorable conditions would be suitable for formal resection; less frequently, more extensive or more formal procedures are unavoidable, but such manipulations almost invariably put the patient's life in jeopardy. It should be remembered that secondary operation as extensive and technical as may be necessary can always be performed on the patient who lives, but the patient who dies leaves nothing on which to work but his gravestone. Much of the rationale of

the preoperative and postoperative care about to be described in connection with the conduct of cases of adynamic ileus applies with equal force to the case with dynamic ileus, and to this the discussion now proceeds.

b. Methods Suitable in Adynamic Ileus: In those cases in which adynamic ileus is engrafted upon a preexisting dynamic ileus the necessity of the removal of any mechanical obstruction to the onward progress of intestinal contents is, of course, axiomatic. After this has been done, however, and from the onset in primary adynamic ileus the fundamental problem in treatment is the restoration of normal tone and motility to the paralyzed intestinal wall.

The reason for the paralysis of the musculature of the intestine in adynamic ileus is by no means clear, and since the rational treatment of any condition depends upon an understanding of the mechanism by which the condition is produced, the treatment of this variety of paralysis is not as obvious as one could wish. Conceivably the fundamental derangement may involve (1) the individual muscle cells of which the musculature of the intestinal wall is composed, (2) the intrinsic nervous regulating system in the wall of the intestine, particularly the plexuses of Auerbach and Meissner, or (3) the extrinsic nervous system, consisting of the vagus and splanchnic nerves.

Little or no evidence has been presented to show that the fundamental derangement occurs within the wall of the intestine itself. Granting, however, that the fundamental derangement in paralytic ileus consists in interference with the blood supply, it is conceivable that local tissue anoxemia, and degenerative changes incidental to vascular insufficiency may play an essential part in the etiology of the condition. If this is so, it would be rational to attempt by some means to reestablish the normal blood supply. Assuming, furthermore, that the fundamental derangement is one of increased intra-intestinal pressure, the fundamental indication would, therefore, be for the evacuation of the intestinal contents, which

may be achieved to some extent, at least, by the performance of one or more enterostomies.

The Rationale of Enterostomy: It must be admitted, of course, that this is not the view of the function of enterostomy most commonly held. Most of those who have written on the subject assume that drainage of toxic material rather than relief of intra-intestinal pressure is the important function of enterostomy, and doubtless this toxic drainage is quite as important as the relief of vascular stasis due to intra-intestinal pressure. In either case, however, the muscle cell profits directly by the procedure of enterostomy.

Enterostomy is a formal operative procedure and one which, though of minor rank in a normal individual, may assume major proportions because of associated toxemia in a patient with adynamic ileus. Undoubtedly certain patients with frank adynamic ileus develop spontaneous intestinal movement under suitable accessory therapy and recover without enterostomy; consequently, the question of whether to enterostomize, and if so when, often gives cause for considerable anxiety. On such a subject it is dangerous to dogmatise, but, according to Lahey[28] in postoperative cases in which a non-mechanical obstruction is diagnosed with reasonable certainty, non-operative measures may usually be employed for about four days with safety. Those obstructions which do not manifest themselves as mechanical will often, according to the teachings of this author,[28] be subsequently found to have been resultant upon local inflammatory processes which eventuate in localized intraperitoneal abscess formation. To attempt to drain such an inflammatory accumulation early, especially when such a condtion is diagnosed merely on suspicion, is distinctly dangerous. Delay until the process has walled itself off and a definite mass becomes palpable is usually entirely safe and the treatment of such a walled-off process, e.g., in the cul-de-sac of Douglas, is thoroughly satisfactory.

The preoperative and postoperative care of the patient about to undergo enterostomy is of importance. In any case,

but especially if the ileus is associated with peritonitis, especial care should be taken to withhold all food and fluid by mouth, and the administration of cathartic drugs should be carefully avoided. Preoperative gastric lavage is of particular importance and should be performed routinely, especially immediately preceding operative manipulations. Lavage performs the function of removing any toxic material which may have found its way by a process of reverse peristalsis into the stomach; it also relieves the stomach and upper part of the intestinal tract of the pressure incident to the presence of this material and by the relief of distention places this portion of the alimentary canal in a favorable condition for the reestablishment of normal intestinal movement. Lavage also tends to prevent regurgitation of stomach contents into the pharynx and mouth during the course of the operative procedure, and inasmuch as this material may be aspirated as the result of such an occurrence, tends to prevent the subsequent development of aspiration pneumonia. For this reason it is also well to leave the stomach tube in place following preoperative gastric lavage so as to provide easy drainage while the patient is on the operating table.

Because of the fact that patients suffering from adynamic ileus are often intensely ill, the selection of a suitable anesthetic agent is a matter of considerable importance. Ordinarily inhalation anesthetics are generally contraindicated not only because of the danger of subsequent aspiration pneumonia, but because of the diminished vital capacity of the lungs which is secondary to abdominal distention, associated elevation of the diaphragm, and consequent compression of the contents of the thorax. Ordinary local analgesia is the method of choice in some cases, but spinal analgesia and especially splanchnic analgesia have much to recommend them inasmuch as both of these methods have a tendency to restore normal motility to the intestinal tube; the mechanism involved will be discussed presently.

Once the abdominal cavity has been invaded, the required operative procedures should be performed with as little

manipulation and as gently as possible. The fundamental object to be achieved is the relief of ileus and preservation of the patient's life. Cosmetic results are of secondary importance, and although secondary operation may be invoked for the subsequent closure of intestinal fistulae, provided that the patient lives, the patient who dies after the performance of a technically brilliant operative procedure is no better off for all the surgeon's skill. Enterostomy in order to be effective must, of course, be performed in such a part of the intestinal tract as to facilitate drainage of intestinal contents from the obstructed area.

Cecostomy is presumably of very little value for this purpose in most cases, because (1) in the primary paralytic variety of ileus there is only local drainage of toxic products, there being no peristalsis to clear the proximal coils, and (2) in the paralytic ileus engrafted upon the obstructve variety the obstruction is usually higher in the small intestine. An enterostomy through an incision in the left hypochondrium into the upper jejunum by the method of Witzel, bringing the enterostomy tube out through a small rent in the omentum after the method of Mayo, has much to recommend it, since by this means the gut is usually drained proximal to a mechanical obstruction if such be the variety, and if the ileus be primarily adynamic the upper portion of the jejunum contains the most virulent toxic material and is in this variety the logical place to perform drainage.

Jejunostomy was advocated for this purpose by Heidenhain as early as 1897,[29] Lennander[30] and Lee and Downs,[31] proceeding on the assumption that toxic products were primarily absorbed from the upper part of the intestinal tract regardless of the site of obstruction, have recently emphasized the value of the method. Haden and Orr,[32] however, found experimentally that, if high intestinal obstruction and jejunostomy were performed in experimental animals both at the same time, the period of survival of these animals was considerably shorter than as though no jejunostomy had been performed at all.

They[33] found, however, that when animals with simple enterostomy received quantities of 1 per cent sodium chloride solution hypodermically the period of survival was much increased, and as the administration of a 1 per cent sodium chloride solution is a relatively simple matter they state that "when any doubt exists concerning the value of enterostomy, it should be performed." Van Beuren,[34] studying the cases of acute ileus submitted to operation in the Presbyterian Hospital in New York during the past twelve years, found a progressive reduction of the mortality rate in the more recent years, which he attributes, first, to the increasing prevalence of early diagnosis, and second, to the use of early enterostomy. He believes that enterostomy performed properly and at the proper time is a procedure of the greatest value. In case it is decided to delay enterostomy, or postoperatively in cases which have received this form of treatment, restoration of motility is still a desideratum.

Drug Therapy in Ileus: The second possible line of attack upon the motor function of the tissue cells of the intestinal musculature consists of stimulation by drugs, the function of which may be either to increase the irritability of the muscle cell or to decrease the threshold for normal stimuli conveyed to the cell by the nervous regulatory system. Among the drugs which have been used for this purpose are eserine, pituitrin, pitocin, choline, acetyl choline, and peristaltin. The relative inefficiency of all of these drugs has previously been discussed in connection with the relief of intestinal distention and gas pains. As Ochsner, Gage, and Cutting[35] have shown, all of these drugs produce a more profound effect on intestines which previously have undergone experimental obstruction than in the normal intestine. However, with the single exception of eserine, none of these therapeutic agents seem to be sufficiently active to be of any particular value in the restitution of normal motility or any degree of motility approaching normal to a gut the seat of ileus, and the drug therapy of paralytic ileus would, therefore, seem to rest on a very precarious basis.

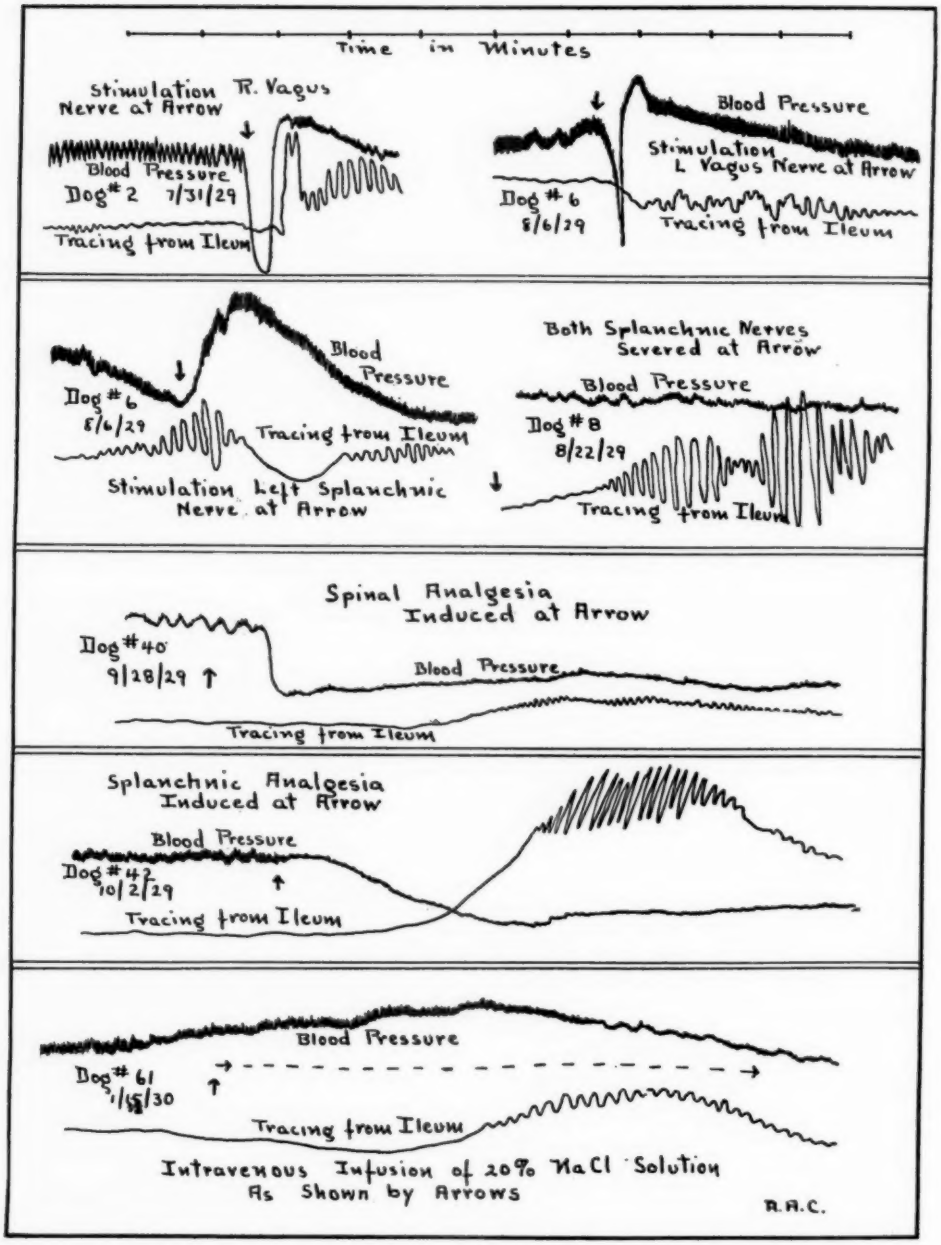

FIG. 76. (Cf. Fig. 37.) Reproduction (greatly reduced) of kymograph tracings showing effect of splanchnic and spinal analgesia and of 20 per cent sodium chloride solution infusions on motor activity of small intestine. At top of figure is a time-marker tracing indicating minutes. With this all of the tracings subsequently reproduced can be compared, since all tracings have been reduced to scale.

Recently various investigators have reported favorably on the use of hypertonic sodium chloride solutions for their effect on intestinal motility. Hughson and Scarff[36] found that intravenous administration of hypertonic sodium chloride solution produced violent peristalsis in animals. This observation was also corroborated by Ross.[37] Ochsner, Gage, and Cutting[35] have found that the administration of a 20 per cent sodium chloride solution is rather effective in producing increases of tone and also increasing the amplitude of intestinal movements. If sodium chloride can be shown to be an efficient intestinal stimulant, it can be made to serve a double purpose, since in ileus the blood chlorides are characteristically decreased and the administration of hypertonic sodium chloride solution would therefore not only increase tone and intestinal movement, but would also be of value in restoring a normal chloride concentration in the blood.

Splanchnic and Spinal Analgesia: Because of the inefficacy of drug therapy designed to stimulate the individual muscle cells or the neuromuscuar mechanism within the wall of the intestine, various attempts have been made to attack the problem from the point of view of the extrinsic regulatory nervous system. On the basis of considerable experimental evi-

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In upper transverse panel are two short tracings indicating effect of vagus nerve stimulation. Note immediate decrease in blood pressure and subsequent stimulation of intestinal motor activity in both cases.

In next transverse panel are two short tracings showing influence of splanchnic nerves on motor activity of intestine. Note cessation of motor activity which follows stimulation of left splanchnic nerve in first tracing and marked stimulatory effect which follows section of two splanchnic nerves in second tracing.

The remaining three transverse panels represent respectively effects of spinal analgesia, splanchnic analgesia, and intravenous infusion of 20 per cent sodium chloride solution. Note depressing action on blood pressure in all cases; note, however, that in the case of spinal analgesia blood-pressure effect is both immediate and profound, whereas in the case of splanchnic analgesia the decrease is gradual and less in degree. Note increase in intestinal tone shown in all cases, and also increase in intestinal motility, which follows after a short latent period. Note that the effect of splanchnic analgesia on intestinal tone and motility is much more profound than that of spinal analgesia, and that the effect of intravenous infusion of 20 per cent sodium chloride solution is intermediate in efficiency.

dence it is generally accepted that the small intestine, at least, is controlled by a dual and antagonistic nerve supply. Nerve fibers going to the gut by way of the vagus nerves are essentially motor or stimulating in their action. The nerve fibers, however, derived from the parasympathetic system and passing to the intestine by way of the splanchnic nerves are inhibitory in their action. This can be demonstrated easily in the intact animal by stimulating the vagus nerve, whereupon intestinal tone and motility are both decreased; by stimulating the splanchnic nerve intestinal tonus and motility are both considerably increased. Conversely, the cutting of the vagi leaves the splanchnic nerves in complete control and the total effect is one of decreased tone and motility, whereas section of the splanchnic nerves leaves the vagus in full control and the resultant effect is an increase in tone and motility. Considering the extrinsic nerve supply of the intestine as of fundamental importance in the development of paralytic ileus, Domenech and others[38] have assumed that in paralytic ileus there is an irritative effect on the splanchnic nerves. The source of this irritative effect in ileus secondary to peritonitis is not far to seek, since it is easy to understand how an inflammatory process of the peritoneum could give rise to reflex splanchnic irritation. It is on this theory, viz., that paralytic ileus is essentially due to splanchnic irritation whether secondary to peritonitis or to some other source of irritation, that the procedures of spinal analgesia and splanchnic analgesia are based. In spinal analgesia the splanchnic fibers are anesthetized at their source as they make their exit from the anterior horn of the various spinal cord segments. In splanchnic analgesia the reflex arc is interrupted after the splanchnic nerves have become fully formed and as they enter into the formation of the greater, lesser, and least splanchnic plexuses in the retroperitoneal space in front of the bodies of the last thoracic and first lumbar vertebrae.

Theoretically, spinal analgesia and splanchnic analgesia should be equally efficient in relieving adynamic ileus, as both

procedures produce a chemical section of the splanchnic nerves. Clinically, spinal analgesia has been used more frequently for this purpose than splanchnic analgesia. Wagner,[39] in 1922, gave the first report of the use of spinal analgesia for the relief of ileus. Since this time numerous observations have been made on the use of the procedure, especially by the French school (Asceriades, Mauclaire, Lapointe, Van Lande, Bosse, and Okinczye, Picot, Guibal and others). Duval,[40] in 1927, was able to collect from the literature 400 cases of acute ileus in which spinal analgesia had been used. Markowitz and Campbell[41] have successfully employed spinal analgesia in treating experimentally produced ileus in dogs. Splanchnic analgesia was suggested by Ochsner, Gage, and Cutting[42] as a preferable method of producing a similar effect, and recently they have presented evidence to show that, as least in experimental animals, splanchnic analgesia is actually more efficient. Rosenstein and Köhler,[43] recognizing the efficiency of splanchnic analgesia, have advocated the use of nicotine by injection into the splanchnic area. They believe that nicotine should be more efficient than novocaine because nicotine has a specific action on sympathetic ganglia. In their experimental investigations they injected nicotine solution directly into the semilunar ganglia and believed that they accomplished by this method a more complete chemical section of the splanchnic nerves than could be achieved by ordinary novocaine solution. The injection of any solution, however, directly into the semilunar ganglia in the human being is quite impossible, the methods available for the induction of splanchnic analgesia being hardly equal to such an accurate localization of drugs. Furthermore, Ochsner, Gage, and Cutting found that nicotine solutions injected in the ordinary way not only produced much less adequate effects than were produced by novocaine solution, but that the blood pressure of experimental animals was reduced to a dangerous degree by the nicotine.

The explanation offered for the superiority of splanchnic over spinal analgesia is that the reflex arc concerned in the

production of ileus is entirely within the splanchnic system. Splanchnic analgesia interrupts this arc in its entirety, presumably producing both sensory and motor effects. Spinal analgesia, on the other hand, interrupts only such portions of the arc as pursue a course within the spinal cord.

More clinical data than at present available must be collected before either the relative or absolute value of these methods can be definitely established. In using splanchnic or spinal analgesia it is essential to realize that success can be expected only in cases in which the intestinal lumen is patent and only in case the therapy is invoked relatively early. The methods are intended not to supplant but to reinforce the treatment of associated toxemia and, inasmuch as neither of these methods seems to affect the large intestine, such treatment should always be supplemented by the insertion of a rectal tube and the administration of an enema. Splanchnic and spinal analgesia when used for the purpose under discussion are performed, of course, not prior to, but subsequent to the performance of enterostomy. A point of particular interest made by Ochsner, Gage, and Cutting[42] is that the effect of splanchnic and spinal analgesia can be absolutely overcome either by epinephrin or ephedrine. Consequently neither of these drugs should be used in conjunction with either method when used for the relief of ileus. Furthermore, since the action of both spinal and splanchnic analgesia is rather fleeting, no hesitation should be felt in repeating either procedure as many times as may be deemed desirable at intervals of an hour or so.

c. The Treatment of the Toxemia of Ileus: Unfortunately, the treatment of the toxemia of ileus is almost entirely symptomatic. Undoubtedly a clearer understanding of the source and method of production of the toxemia would simplify the treatment considerably, but until such an understanding is gained the best that can be done is to attempt to combat the manifestations of perverted metabolism insofar as they are known.

1. *The Use of Water, Sodium Chloride, and Dextrose:* The dehydration and hypochloremia of intestinal obstruction are conveniently treated together. The obvious treatment is the administration of quantities of sodium chloride solution. Inasmuch as the administration of anything by mouth is strictly contraindicated in ileus, fluids must be administered either rectally, subcutaneously, or intravenously. Ileus, constituting as it does a grave condition and the need for fluid and sodium chloride being acute, either the subcutaneous or the intravenous route is almost universally adopted. Orr and Haden[33] recommend the intravenous administration of 500 c.c. of a 5 per cent sodium chloride solution intravenously in all cases subjected to operative procedures. Eliason[44] recommends a 5 per cent glucose solution in normal salt solution, McVicar and Weir[45] a 10 per cent glucose solution in 1 per cent sodium chloride solution.

The addition of hypertonic dextrose solution to solutions for intravenous administration is recommended because of the effect on diuresis. As has been previously mentioned, patients suffering from ileus characteristically show the phenomenon of nitrogen retention. Promotion of diuresis is, therefore, presumably of value in removing the excess of nitrogenous waste products from the blood stream. Postoperatively, and indeed at all times, patients with ileus should be subjected to blood chemistry examinations at frequent intervals, at least twice daily, to determine the efficiency of the treatment.

Careful hemoglobin estimations should be performed to determine whether or not blood concentration is being sufficiently combated. Plasma chloride determinations should be made to determine the presence or absence of hypochloremia; non-protein nitrogen determinations to determine the presence or absence of nitrogen retention, and estimations of the carbon dioxide combining power of the plasma to determine the status of the acid-base balance. It would be futile to attempt to state either the amount of fluid or the composition of fluid suitable for all cases. Most cases require at least from 3 to 5 l.

of fluid per twenty-four hours. All patients need sodium chloride either in normal or hypertonic solution, and probably most patients need dextrose in from 5 to 10 per cent solution. The requirements of individual patients with regard to these substances, however, vary, and it is only by the aid of accurate and frequent laboratory findings that the proper amount of the substances can be determined with accuracy.

2. *The Use of Gastric Lavage and External Heat:* Gastric lavage has been mentioned as a valuable preoperative procedure, but frequent lavage of the stomach postoperatively is almost equally important both in the relief of toxemia and in the promotion of the patient's comfort. Another measure of great value in promoting the patient's comfort, and possibly in the actual treatment of the condition, is the application of heat to the abdomen. Muller[46] has ventured the hypothesis that in paralytic ileus the decrease of movement in the intestinal tract is associated inseparably with increase in secretion. He believes that the application of heat to the abdomen produces a dilatation of peripheral vessels of the abdomen which is associated with a contraction of the splanchnic vessels by a mechanism of visceral splanchnic balance. The contraction of the splanchnic vessels not only diminishes intestinal secretion, but also increases intestinal motility.

3. *The Use of Perfringens Antitoxin:* A number of attempts have been made in recent years to incriminate the Bacillus welchii group of organisms in the production of the toxemia of intestinal obstruction. Williams,[47] in 1926, fostered this theory. He found that Bacillus welchii grew best in a slightly acid or neutral medium, and considered that such a medium was found in the upper, rather than in the lower, portion of the intestinal tract. He made use of immune serum in the clinical treatment of certain cases and reported favorable results. Experimentally, Stabins and Kennedy,[48] McIver, White, and Lawson[49] found that whereas the Bacillus welchii was present in small numbers in the normal intestinal canal, they increased very remarkably in the presence of intestinal obstruction.

Bower and Clark,[50] Morton and Scabins,[51] following the lead of the previously named authors, have reported favorable results with antitoxin, the former in clinical and the latter in experimental cases. Oughterson and Powers,[52] and Owings and McIntosh[53] have, however, been unable to confirm these findings, the former making use of antitoxin and serum obtained by the active immunization of animals against the Welch bacillus, the latter authors employing the bacillus perfringens antitoxin.

VII. Prognosis in Ileus

The prognosis of a well-established case of intestinal obstruction is invariably grave. When associated with peritonitis, the prognosis is even more grave. Of chief prognostic importance is the time elapsing between the onset of the obstruction and the performance of operative procedures. Van Beuren[54] has stated this succinctly in his aphorism that the longer a patient with intestinal obstruction lives prior to operation, the sooner he dies afterwards.

Miller[55] states the same truth in a slightly different manner by affirming that as a general rule the mortality rises approximately 1 per cent for each hour of delay in the relief of obstruction. Miller, reviewing a series of 343 cases, found that the mortality rate when the patient was subjected to operative procedures within twelve hours of the onset of symptoms was only 29.4 per cent. Within twenty-four hours this percentage had increased to 42.9 per cent; within thirty-six hours to 50 per cent; within forty-eight hours to 59.6 per cent; within seventy-two hours to 63.4 per cent; within ninety-six hours to 72.8 per cent, and in cases which had persisted for more than ninety-six hours an 84 per cent mortality was observed.

Brill,[56] in a series of 83 cases, found that the mortality rate was nil when patients were subjected to operation within twelve hours of the onset of symptoms. When such operations were performed in periods between twelve and twenty-four

hours, there was a 12.5 per cent mortality, and between twenty-four and forty-eight hours a 61.1 per cent mortality.

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AUTHOR INDEX TO VOLUME XIV

Alessandri, Roberto, 68
Alvarez, Walter C., 385

Bierman, William, 626
Bloodgood, Joseph Colt, 89
Bourne, Wesley, 653

Christopher, Frederick, 652
Coffey, Robert C., 161
Coley, William B., 605
Collins, Clifford U., 449
Crile, George W., 288
Cutting, R. A., 507

Deaver, John B., 276
Doran, William G., 442

Eisenstaedt, J. S., 477

Faulkner, William B., Jr., 646
Finney, J. M. T., 149
Finney, J. M. T., Jr., 149
Flothow, P. G., 591

Galloway, Charles Edwin, 643
Graham, Evarts A., 382
Gutierrez, R., 657

Halperin, J., 637
Harris, Augustus, 480
Hoffman, Frederick L., 229
Horsley, J. Shelton, 264
Howell, W. B., 692

Jarcho, Julius, 419

Kantor, John L., 620
King, Walter E., 460

La Rochelle, F. D., 452
Leriche, René, 55
Lilienthal, Howard, 356
Lynch, Jerome M., 624

McDougall, T. G., 477
Maliniak, Jacques W., 483
Marbury, W. B., 499
Mathieu, Albert, 634
Mayo, William J., 329
Moure, Paul, 298, 309

Peck, Willis S., 472
Phaneuf, Louis E., 428
Potter, Eugene B., 472

Raginsky, B. B., 653
Reid, Mont R., 17
Ribas y Ribas, E., 215, 225
Robertson, Hugh, 395
Roeder, C. A., 454
Royster, Hubert A., 79

Schmier, Adolph A., 431
Shelley, Harold J., 631
Simpson, Walter M., 502

Trias-Pujol, Joaquin, 313, 323

Updegraff, Howard L., 492

Van Buren, F. A., 458

Welton, T. S., 501, 689, 691
Wilkie, David P. D., 340

Yeomans, Frank C., 489

SUBJECT INDEX TO VOLUME XIV

(B.B.) = Bookshelf Browsing. (Bi.B.) = Biographical Brevities. (B.R.) = Book Review. (C.) = Correspondence. (E.) = Editorial.

Abdomen, injured, 395
Abdominal pain, 385
Aesculapius, a chat with (B.B.), 692
Anesthesia, 288
  spinal, carbon dioxide as adjunct to, 454
Apparatus to reduce shoulder dislocations, 452
Appendicitis (E.), 699
Appreciation of Professor Rudolph Matas, 1
Arterectomy, experimental and clinical basis for, in treatment of localized arterial obliterations, 55
Avertin, effect of, upon normal and impaired liver, 653

Bladder, rupture of, 477
Book Reviews:
  Aids to Surgical Anatomy (Hunter), 698
  Artificial Sunlight (Luckiesh), 699
  Asthma and Hay Fever (Coca), 506
  Cutaneous x-ray and Radium Therapy (Hazen), 700
  Demonstrations of Physical Signs in Clinical Surgery (Bailey), 506
  Diagnosis and Treatment of Venereal Diseases in General Practice (Harrison), 702
  Diseases of the Gums and Oral Mucous Membrane (Goadby), 698
  Encephalitis Lethargica (von Economo), 701
  Fractures of the Jaws (Ivy and Curtis), 699
  Great Physician, Osler (Reid), 505
  Infant Welfare Movement in the 18th Century (Caulfield), 701
  Lehrbuch der Röntgenstereoskopie (Cone and Barth), 699
  Maligne Pharynx und Larynxtumoren (Zuppinger), 699
  Modern Proctology (Pruitt), 698
  Normale und pathologische Funktionen der Verdauungsorgane im Röntgenbild (Becker and Oppenheimer), 700
  Renal Lesion in Bright's Disease (Addis and Oliver), 697
  Röntgendiagnostik der Gallenblase (Eisler and Kopstein), 505
  Surgical Clinics of North America, 505
  Theory of Obstetrics (De Garis), 506
  Urological Roentgenology (Young and Waters), 698
Breast, cancer of, 276
  male, unilateral idiopathic hypertrophy of, 652

Cancer of breast, 276
  control, some thoughts on the problem of, 605
  in North American negro, 229
  of rectum and rectosigmoid, 161
  of stomach with special reference to its incidence, diagnosis and treatment, 264
Carbon dioxide as adjunct to spinal anesthesia, 454
Cephalic and pelvic diameters, roentgenographic measurement of, 419
Cervical ribs, 449
Cesarean section, staphylococcemia following, 637
Cysts, hydatid, fistulas following marsupialization of, their radiographic examination, 313

Dislocations, shoulder, apparatus to reduce, 452

Editorials, *a*, 499, 689
Experimental and clinical basis for arterectomy in treatment of localized arterial obliterations, 55

Fibroid, uterine, separated from its pedicle, 631
Fistulae, bronchial, observations on reactions of, to acute infections of upper respiratory tract, 382
  following marsupialization of hydatid cysts: their radiographic examination, 313
Focal infection, 643
Fractures, upper extremity, hospital care of, 442

Gas bacillus infection in civil life, 460
Granulating areas, extensive, treatment of, 472

Halstead thirty-six years ago, 89
Hemorrhoidectomy by means of high frequency current, 626
Hemorrhoids, injection treatment of, 489
  medical aspect, 620
  surgical treatment, 624
High frequency current, hemorrhoidectomy by means of, 626
Hospital care of upper extremity fractures, 442
Hypertrophic tuberculosis of the ileum with multiple lesions, 149
Hypertrophy, unilateral idiopathic, of male breast, 652
Hysterosalpingography by means of iodized rapeseed oil, 634

Ileum, hypertrophic tuberculosis of, with multiple lesions, 149
Implant, cartilage, problem of, 492
Infection, focal, 643
  gas bacillus, in civil life, 460
  prevention of, in prostatectomy, 480
Injection treatment of hemorrhoids, 489
Injections, diagnostic and therapeutic, of the sympathetic nerves, 591
Injured abdomen, 395
Intestinal obstruction from congenital deformity of small intestine, 215
Inversion, puerperal, of uterus, 428
Is the blood flow in varicose veins reversed? 431

Kidney, horseshoe, clinical management of, 657

Lesions, vascular, that have been taught and emphasized by Professor Matas, report of, 17
Lest we forget (E.), 689
Liver, normal and impaired, effect of avertin upon, 653

Matas, Professor Rudolph, appreciation of, 1

Negro, North American, cancer in, 229
Nélaton's line, (Bi.B.), 691
Nerves, sympathetic, diagnostic and therapeutic injections of, 591

Observations on reaction of bronchial fistulae to acute infections of upper respiratory tract, 382
Obstruction, intestinal, from congenital deformity of small intestine, 215
Operative treatment of phthisis, the surgeon's moral obligation, 356

Pelvic and cephalic diameters, roentgenographic measurement of, 419
Personal experience in vascular surgery, 79
Phthisis, operative treatment of, the surgeon's moral obligation, 356
Place of the senses in the development of science, 329
Plastic surgery and the specialists, 483
Pneumothorax, diagnostic, 696
Preoperative and postoperative treatment, principles of, 509, 703
Prevention of infections in prostatectomy, 480
Principles of preoperative and postoperative treatment, 509, 703
Problem of cartilage implant, 492
Prostatectomy, prevention of infections in, 480
Puerperal inversion of uterus, 428

Raynaud's disease and thromboangiitic gangrene of upper extremities treated by resection of sympathetic ganglia and trunk, 68
Rectosigmoid, rectum and, cancer of, 161
Rectum and rectosigmoid, cancer of, 161
Report of vascular lesions that have been taught and emphasized by Professor Matas, 17
Respiratory tract, upper, observations on reaction of bronchial fistulae to acute infections of, 382
Rhinoplasty, total, 298
Ribs, cervical, 449
Roentgenographic measurement of pelvic and cephalic diameters, 419
Rupture of the bladder, 477
of uterus following cesarean section, 458

Science, place of senses in development of, 329
Splenectomy: its indications and technique, 340
Staphylococcemia following cesarean section, 637
Stensen's duct (Bi.B.), 501
Stomach, cancer of, with special reference to its incidence, diagnosis and treatment, 264
Surgery, plastic, and the specialists, 483
Sympathetic nerves, diagnostic and therapeutic injections of, 591

Thromboangiitic gangrene, Raynaud's disease and, of upper extremities treated by resection of sympathetic ganglia and trunk, 68
Total rhinoplasty, 298
Treatment of extensive granulating areas, 472
preoperative and postoperative, principles of, 509, 703
Tuberculosis, hypertrophic, of ileum with multiple lesions, 149

Uterine fibroid separated from its pedicle, 631
Uterus, puerperal inversion of, 428
rupture of, following cesarean section, 458

Varicose veins, is the blood flow reversed in? 431
Vascular surgery, personal experience in, 79

Warthin, Aldred Scott (B.B.), 502